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**New York State**
**Department of Labor**

**Division of Industrial Hygiene**
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*For additional material on this subject consult New York State Department of Labor Special Bulletin, Carbon Monoxide Poisoning in Industry, 1930; Special Bulletin No. 194, Carbon Monoxide Poisoning and Its Prevention, 1938 (now in process of revision); files of "Monthly Review," Division of Industrial Hygiene and Safety Standards; Industrial Bulletin prior to 1946.*

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# CARBON MONOXIDE POISONING

May R. Mayers M.D.

Under normal conditions, when a person breathes fresh air, the coloring matter of his red blood cells—known as hemoglobin—combines with the oxygen which he breathes, and carries it from his lungs to all parts of his body, thus providing nourishment for his tissue cells. Unless the oxygen is brought to them in this way, his tissues suffer from oxygen-want exactly as they would if there were no oxygen available for him to breathe. The mere intake of oxygen into the lungs during inspiration is thus, in itself, quite insufficient. In order that it may be utilized, it must combine with the hemoglobin of the red cells and be carried through the body in the circulating blood.

Carbon monoxide is an odorless and colorless gas which is incapable of causing direct irritation to the lungs or the upper respiratory passages. It has a peculiar affinity for hemoglobin, however—approximately 300 times as great as is the affinity of oxygen for hemoglobin. As a result, when both are present at the same time in the air breathed, the carbon monoxide combines far more readily with hemoglobin than does the oxygen, and even tends to displace such oxygen as was already in combination with it. This causes what is known as an anoxemia—a condition in which there is an inadequate oxygen supply to tissue cells, with a corresponding degree of relative tissue asphyxia.

When carbon monoxide displaces oxygen from the hemoglobin molecule, it does so molecule for molecule—that is to say, one molecule of carbon monoxide displaces one molecule of oxygen. The whole reaction is in the nature of a reversible mass action, depending for its direction upon the relative tension of the two gases.

## Rate of Absorption of Carbon Monoxide Gas

It has been estimated that about one-half the equilibrium is established in the first hour, and about three-fourths in the first two hours. Thus if an individual is exposed to an atmosphere containing a given amount of carbon monoxide, his body will, within the first two hours, absorb three-fourths of the total amount which can possibly be absorbed.

The concentration of carbon monoxide in the air breathed; the duration of exposure; the temperature, humidity and air movement

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in the workroom; the size, age and activity of the individual exposed, as well as his metabolic rate, and the amount of hemoglobin which he happens to possess at the time of his exposure, are all important factors governing the rate of absorption of the gas.

The importance of *physical activity* in increasing the rate of absorption of carbon monoxide gas—even to the point of being the precipitating cause in the production of actual asphyxia—is not sufficiently appreciated. On the one hand, by increasing the respiratory rate, exercise increases the rate of inhalation and absorption of the gas. With an increased absorption comes a corresponding reduction in the amount of oxygen available to the body for the nourishment of tissue cells. On the other hand, physical exercise immediately increases the oxygen requirements of the body—especially of those groups of muscles most directly involved. It may readily be seen, therefore, that in an individual whose available oxygen supply is already reduced because of carbon monoxide absorption, even moderately increased activity may call for a supply of oxygen in excess of that which is then available. In such event, acute asphyxia ensues.

A further factor must not be lost sight of. Physiologists have shown that during strenuous exercise, or in any other condition resulting in an anoxemia of tissue cells there is, among other things, an accumulation of lactic acid. During the period when this is being oxidized, for purposes of elimination from the body, a supply of oxygen considerably in excess of the normal is required. The demand for oxygen thus continues to be abnormally great not only during the actual period of exercise, but for some time thereafter.

In persons already in a state of anoxemia due to carbon monoxide absorption one may, therefore, readily see how exercise could seriously aggravate an already existing oxygen deficiency. Haldane has called attention to the fact that a worker at rest exposed to carbon monoxide gas can have half his blood saturated with carbon monoxide without losing consciousness. If he is at work, however, unconsciousness results when only one-third of his blood has become saturated with the gas. All this has been graphically illustrated, time and again, in the case of persons who have accidentally awakened to find themselves in a room filled with illuminating gas. If such a person lies perfectly still until he is rescued, he may retain consciousness, whereas any movement on his part to reach the door may render him unconscious. Similarly in workers exposed to carbon monoxide gas, it has been observed that a rush assignment or sudden exertion preparatory to going home, or for whatever reason, may most unexpectedly precipitate an attack of asphyxia, even

when the actual carbon monoxide content of the air at the moment is not excessive.

The potentialities of the situation for a given worker at any given time may perhaps best be appraised, if thought of as the resultant of the factors making for tissue anoxemia (i.e. carbon monoxide absorption and exercise—the latter in its double role of increasing the rate of carbon monoxide absorption and acting independently to produce further anoxemia associated with fatigue), and those factors determining the supply of oxygen which is available to him for immediate combustion (i.e. the oxygen content of his blood and the oxygen in the air breathed).

## The Elimination of Carbon Monoxide From the Body

On breathing fresh air such carbon monoxide as was previously absorbed is eliminated from the body at the rate of 30 to 60 per cent reduction of blood concentration per hour. While the elimination of carbon monoxide from the blood leaves the individual red cells substantially unchanged, and they are once again able to carry oxygen to the tissues in a normal manner, body cells which have in the meantime been either partially or completely destroyed, because of lack of oxygen, cannot always be regenerated. This occurs particularly in the nervous system, and accounts for many of the serious sequelae of carbon monoxide poisoning, some of which are permanent and others of very long duration. Yant in his recent pamphlet, *"Studies in Asphyxia,"* calls attention to how few persons who have become moribund as a result of carbon monoxide poisoning seem to recover, even though all of the carbon monoxide is very rapidly removed from their bodies by artificial methods of resuscitation.

When blood samples are taken for purposes of diagnosis, they must be taken as soon as possible after exposure, because within approximately four hours after removal from the gas practically all of the carbon monoxide will usually have been eliminated from the body.

## Differences in Individual Susceptibility

Differences in individual susceptibility to carbon monoxide gas are very striking and should never be lost sight of. These are in part at least, due to marked differences in the ability of individuals to maintain a normal oxygen supply to their tissues despite their absorption of the gas. Various compensating mechanisms come into play when the body is being faced with an oxygen deficiency, and it would appear that in the case of the less susceptible individuals

these mechanisms are set in motion more promptly, and are in the long run more effective than they are in other persons.

Where oxygen deficiency is threatened as a result of exposure to carbon monoxide gas—a certain amount of the oxygen having been displaced from the red cells by the carbon monoxide—two principal compensatory mechanisms normally come into play:

(1) As an immediate emergency measure, the spleen begins to contract, and large numbers of normal red cells are hurriedly thrown into the circulation—at once increasing the available amount of normally functioning hemoglobin.

(2) After a period of adjustment, there may be an increase in the production of red cells by the blood-forming organs, (particularly the bone marrow), resulting in an increase in total hemoglobin, and thus an increase in the oxygen carrying capacity of the circulating blood.

If the individual exposed can compensate adequately along these lines for whatever hemoglobin has been rendered functionless—and some can do this if the concentration of carbon monoxide is not too great or exposure too long—his tissues will receive approximately their normal oxygen supply, and he may succeed in adapting himself tolerably well to his abnormal environment.

There are individuals, however, whose blood-forming organs cannot adequately make the necessary adjustments, or can do so only for a limited period of time. Prolonged exposure to carbon monoxide gas in these cases ultimately results in decreased cell production with a diminishing tolerance to the gas.

It is only by careful and intelligent medical supervision of workers exposed, that susceptible individuals may be weeded out before irreparable damage to their health has been done.

## Age

In general, smaller persons tend to be more susceptible to carbon monoxide gas than are larger ones, because the rate of respiration in the former group is on the whole more rapid—resulting in a tendency to greater absorption of the gas in a given period of time. For this reason minors are peculiarly susceptible to carbon monoxide gas, and should not be permitted to work in industries where there are possibilities of exposure. The tendency to an increased susceptibility among old people is discussed below under "Co-existing Disease."

## Sex

There is some difference of opinion as to whether women are more or less susceptible to carbon monoxide gas than are men. Cases

have been reported in which women were sole survivors in group asphyxia. However, conclusive data on this point is lacking. Luden has reported disturbances in menstruation following prolonged exposure to carbon monoxide gas where there appeared to be every reason to believe that there was causal relation. Glaister and others have called attention to the danger to pregnant women since concentrations of the gas, too small to be injurious to them, have been known to cause death of the fetus. This would certainly warrant the exclusion of pregnant women from exposure to carbon monoxide gas in industry.

### Co-existing Disease

It has been pointed out that persons with abnormally high respiratory rates whether due to increased basal metabolism, cardiac disease, or other causes, are poor subjects for exposure to carbon monoxide gas. Workers who are anemic or are suffering from other blood diseases should not be exposed to the gas. Back believes that individuals suffering from focal infections as well as those having pulmonary or cardiac disease are, generally speaking, more susceptible to carbon monoxide poisoning than are others. Clinical experience has led to the belief that old people are unduly susceptible to the gas and recover very slowly from symptons of "gassing." The relatively higher incidence of co-existing chronic disease among old people—particularly of the cardio-vascular system—may, perhaps, account for these observations.

### Exposure to Varying Concentrations of Carbon Monoxide Gas

The injurious effects of exposure to varying concentrations may be seen in the following table prepared by Sayers and Yant of the Bureau of Mines:

| *Symptoms* | *Percentage of Blood Saturation* |
|---|---|
| No symptoms | 0–10 |
| Tightness across forehead; possible slight headache; dilation of cutaneous blood vessels | 10–20 |
| Headache; throbbing in temples | 20–30 |
| Severe headache; weakness; dizziness; dimness of vision; nausea and vomiting; collapse | 30–40 |
| Same as previous item with more possibility of collapse and syncope, increased respiration and pulse | 40–50 |
| Syncope; increased respiration and pulse; coma with intermittent convulsions; Cheyne-Stoke's respiration | 50–60 |
| Coma with intermittent convulsions; depressed heart action and respiration; possible death | 60–70 |
| Weak pulse and slowed respiration; respiratory failure and death | 70–80 |

Henderson's experiments in connection with the building of the Holland vehicular tunnel showed that subjects who were exposed

for two hours or more to concentrations of four parts of carbon monoxide per 10,000 suffered distinct loss of efficiency with symptoms of headache, nausca, etc. When exposed to two parts per 10,000 for six hours, there were slight symptoms at the end of the experiment. Those exposed to three parts per 10,000 showed the same symptoms in two and one-half to three hours. It was concluded that no deleterious effects followed exposure to three parts per 10,000 for one hour. Exercise, as well as high temperatures and humidities, were shown to cause earlier appearance of symptoms, and a rise in the body temperature, pulse rate and respiration. The concentration of carbon monoxide which was adopted as offering a good margin of safety in the vehicular tunnel was four parts per 10,000 on the assumption that it would never take more than 45 minutes to drive through it, and usually less than half an hour.

Henderson has suggested the following formulae for calculating the psysiological effects to be looked for in short exposures to carbon monoxide gas—not exceeding a very few hours:

| | | |
|---|---|---|
| Time x concentration = | 3. | No perceptible effect. |
| Time x concentration = | 6. | A just perceptible effect. |
| Time x concentration = | 9. | Headache and nausea. |
| Time x concentration = | 15. | Dangerous. |

## Prolonged Exposure to Relatively Low Concentrations*

Sayers and Yant have pointed out that prolonged exposure to the lower concentrations of carbon monoxide gas produce more severe and more lasting symptoms than do short exposures to the high concentrations.

It has been generally accepted that carbon monoxide is not a cumulative poison in the sense that lead is, since it is not stored in the body even in small amounts. In persons who are regularly exposed to the gas during working hours, however, its continued absorption produces a state of daily recurrent anoxemia—or lack of normal oxygen supply to the body—of varying degrees of intensity. While the oxygen deficiency with which such workers must contend while at work eventually tends to clear up when they go home at night and get out into the fresh air, distressing symptoms of one kind or another may continue for many hours after all carbon monoxide has been eliminated from the body. The severe pounding headache, for example—due to edema (swelling) of the brain—may continue all night, and even into the next day, sometimes making it impossible to go to work the next morning.

* Concentrations in excess of one part of carbon monoxide gas per 10,000 parts of air, but below that required to cause acute carbon monoxide poisoning or asphyxia.

This may be associated with general irritability, insomnia, dizziness, gastro-intestinal upset, and a sense of weakness and general lassitude. Indeed many of these symptoms may be present even in the absence of the headache, and may be chronic complaints of these workers—regardless of the specific extent of exposure on any given day.

On examination of the blood, a secondary anemia is not uncommonly found among them. On the other hand, among those who are capable of compensating more adequately for the oxygen deficiency, there may be found an abnormal increase in the number of red blood cells, and the amount of hemoglobin in their blood.

When one studies the profound functional disturbances—both physiological and bio-chemical—initiated in the body by a state of anoxemia quite inadequate in intensity to produce unconsciousness, one is impressed with the potential dangers of this type of exposure. The extent to which lasting injury may be anticipated would seem to be dependent upon the degree of the anoxemia and its duration; the vulnerability of tissue cells to oxygen deficiency *in the given individual*—particularly in terms of pre-existing disease and inherited tendencies—and the powers of tissue regeneration of which he is capable. While the many factors involved obviously permit of enormous individual variations, constantly recurring anoxemia, resulting from continued exposure to carbon monoxide gas of a degree sufficient to produce symptoms even if not sufficient to cause acute asphyxia, would seem to be a real menace to health.

### Repeated Attacks of Acute Poisoning

Repeated exposures to carbon monoxide gas—particularly repeated attacks of acute carbon monoxide poisoning or actual asphyxia—have been shown to result in a progressively increasing susceptibility to the gas, as well as in cumulative tissue damage.

Experimental work on animals directed toward an evaluation of this situation, has shown that this is particularly true with regard to the central nervous system. Farraro and Morrison, in their experiments with rabbits, for example, found that the pathological changes were apparently greater with each successive exposure. Indeed, after a number of exposures, injury to the nervous system appeared to be as great for a short exposure to a given concentration of the gas as was produced by a very much longer exposure to the same concentration, the first time.

### Exposure to Mixed Gases Containing Carbon Monoxide

Furthermore, in attempting to appraise the health hazards associated with exposure to carbon monoxide gas in indusry—particu-

larly the effects of prolonged exposure to concentrations insufficient in amount to cause acute asphyxia—one cannot overlook the fact that such workers may be exposed to a combination of gases in which carbon monoxide is merely one of many potentially toxic constituents.

Henderson has shown, very interestingly, that illuminating gas even in relatively low concentrations will kill a fragment of chick brain, even though the specimen grew very satisfactorily in an atmosphere of pure carbon monoxide gas. It has also been shown that exposure to illuminating gas kills insects which have no hemoglobin, and are thus immune to carbon monoxide poisoning. Further experiments on animals have demonstrated that when exposed to illuminating gas they cannot withstand as large a displacement of hemoglobin from their red cells as when pure carbon monoxide is used. Thus, in the presence of illuminating gas, for example, they invariably die when their blood becomes 50 per cent saturated with carbon monoxide, while such animals have survived a blood saturation of even 80 per cent when chemically pure carbon monoxide was used.

It has already been pointed out that exposure to coal gas and coal tar distillates appears to be more dangerous than is exposure to pure carbon monoxide gas in equal concentrations. This is believed to be due to their benzol content.

Rudolf Hofer demonstrated experimentally that the toxic action of mixed gases is far greater than that of each individual gas when used alone. His very important findings may be briefly summarized as follows:

> "1—Combination tests with carbon monoxide and hydrogen sulfide show that hydrogen sulfide in concentrations of 0.04 per cent and carbon monoxide in concentrations of 0.5 per cent, which do not have a fatal effect when inhaled for a period of 10 minutes, are found to be deadly when inhaled simultaneously for the same length of time. When the experimental animal was exposed for a longer period than this, one-half the fatal concentration of each gas was sufficient to cause death when given in combination.
>
> "2—The combination tests with carbon monoxide and hydrocyanic acid show that even a combination of 0.1 per cent carbon monoxide and 0.0018 per cent hydrocyanic acid induces severe paralysis (in lateral position). The same degree of action is not achieved in separate inhalations until the cat has inhaled 0.25—0.3 per cent carbon monoxide (i.e., a concentration two and one-half to three times as great) and 0.0045 per cent hydrocyanic acid (i.e., a concentration more than twice the above)."

Where there is exposure to high concentrations of such carbon monoxide-containing gases as illuminating gas, or the exhaust gas from automobiles, the asphyxia resulting from the severe and rapid anoxemia produced by the carbon monoxide so dominates the clinical and pathological picture as to relegate everything else to a position of relative insignificance. However, where there is prolonged intermittent exposure to small amounts of these gases, the common practice of attempting to appraise and interpret the clinical pictures presented by workers so exposed, *solely* in terms of the degree of anoxemia produced by the carbon monoxide which they inhaled (as though this were the only gas present) would seem to be somewhat misdirected.

There is need for further study both on animals in the laboratory, and on workers in industry, of the toxicological effects of mixed gases in which carbon monoxide is one of the constituents. This is especially necessary for prolonged exposure to relatively small amounts of these gases—amounts sufficient to cause clinical symptoms, and loss of time on the part of workers, and yet insufficient to cause acute asphyxia. Until more data is available along these lines there is need for caution in the interpretation of illnesses, believed rightly or wrongly by workers to be due to such exposure.

## Carbon Monoxide As a Tissue Poison

One cannot close a discussion of the toxicology of carbon monoxide gas without pointing out that while it is the generally accepted view—particularly in this country—that this gas is in no sense a tissue poison, this is still a matter which has not been finally settled. There are still those both in this country and abroad, who believe that while its *principal* action is undoubtedly the production of an anoxemia through displacing oxygen from the hemoglobin of the red cells, it may in addition have other physiological effects not yet fully understood. The reasons for differences of opinion on so important a matter are too technical for discussion in a Bulletin of this type.

# THE EFFECT OF CHEMICALLY PURE CARBON MONOXIDE, ILLUMINATING GAS, AND AUTOMOBILE EXHAUST GAS UPON THE FRAGILITY OF THE RED BLOOD CELLS

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To investigators interested in the subject of carbon monoxide poisoning, the question of the relative toxicity of carbon monoxide in its pure state and of carbon monoxide as found in such substances as illuminating gas and the exhaust gas from automobiles is one of considerable importance.

Henderson and Haggard,[1] who conducted a series of interesting experiments along these lines, have shown that illuminating gas, even in relatively low concentration, will kill a fragment of chick brain, though the latter has been shown to grow very satisfactorily in an atmosphere of pure carbon monoxide. They have also shown that exposure to illuminating gas will kill insects which have no hemoglobin and which are known to be immune to carbon monoxide poisoning. Further experiments by the same investigators[2] have shown that when animals are exposed to illuminating gas or to exhaust gas from a car using coal distillate, the carbon monoxide contained in these gases appears to be far more toxic to them than is the same concentration of pure carbon monoxide. Indeed, in the presence of these gases, if their blood becomes, respectively, 70 per cent and 60 per cent saturated with carbon monoxide, the animals invariably die, while even a blood saturation of 80 per cent, was *survived* by the *same animals* when chemically pure carbon monoxide was used. Henderson and Haggard consider that while carbon monoxide is undoubtedly the chief toxic element in these gas mixtures, as much as 25 per cent of their toxicity is probably due to other substances.

Rudolf Hofer[3] conducted a careful experimental study for the purpose of determining the toxic action of mixed gases, as compared

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with the toxicities of each individual gas. His findings are extremely pertinent to the present discussion, and may be briefly summarized as follows:

> 1. Combination tests with carbon monoxide and hydrogen sulphide show that hydrogen sulphide in concentrations of 0.04 vol. % and carbon monoxide in concentrations of 0.5 vol. %, which do not have a fatal effect when inhaled for a period of 10 minutes, are found to be deadly when inhaled simultaneously for the same length of time. When the experimental animal was exposed for a longer period than this, one-half the fatal concentration of each gas was sufficient to cause death when given in combination.
>
> 2. The combination tests with carbon monoxide and hydrocyanic acid show that even a combination of 0.1 vol. % carbon monoxide and 0.0018 vol. % hydrocyanic acid induces severe paralysis. The same degree of action is not achieved in separate inhalations until the cat has inhaled 0.25–0.3 vol. % carbon monoxide (i. e., a concentration two and one-half to three times as great) and 0.0045 vol. % hydrocyanic acid (i. e., a concentration more than twice the above).

It would seem from these experiments that a mixture of carbon monoxide with one or more toxic gases is more toxic than is the sum of the toxicities of the individual gases.

Since large numbers of workers are exposed to illuminating gas and exhaust gas rather than to pure carbon monoxide, it becomes a matter of more than theoretical interest to arrive at a proper understanding of the precise differences in their relative toxicities, in terms of physiologic and biochemical reactions upon those exposed. The present series of *in vitro* experiments was undertaken with a view to acquiring information upon only one small phase of the question—namely, the relative power of chemically pure carbon monoxide, illuminating gas, and exhaust gas to hemolyze the red blood cells. To this end, normal blood was exposed to all three substances to the point of saturation, and the fragility of the red blood cells was determined as described in the following report.

## Experimental Study

### *1. Fragility of Untreated Red Blood Cells*

*Procedure*—Each 15 c.c. of blood was collected in a tube containing the residue of 1 c.c. of sodium citrate (8 per cent. solution). Then 0.05 c.c. of whole blood was pipetted into 2 c.c. of sodium chloride solution (concentrations ranging from 0.25 to 0.6 per cent)

and mixed carefully. Examination for hemolysis was made after two hours and after twenty-four hours. Duplicate determinations were done on each blood.

*Results*—The findings on three bloods (Table 1) show that complete hemolysis occurred in sodium chloride concentrations between 0.25 and 0.33 per cent, and partial hemolysis in concentrations between 0.34 and 0.43 per cent:

TABLE 1.—DEGREE OF HEMOLYSIS: UNTREATED RED BLOOD CELLS

| BLOOD SAMPLE | COMPLETE HEMOLYSIS | | PARTIAL HEMOLYSIS | | NO HEMOLYSIS | |
|---|---|---|---|---|---|---|
| | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests |
| 1 | 0.25–0.33 | 9 | 0.34–0.43 | 10 | 0.44–0.60 | 17 |
| 2 | 0.25–0.33 | 9 | 0.34–0.43 | 10 | 0.44–0.60 | 17 |
| 3 | 0.25–0.32 | 8 | 0.33–0.43 | 11 | 0.44–0.60 | 17 |

### 2. *Fragility of Aerated Red Blood Cells*

*Procedure*—Air which had been washed through sulphuric acid was bubbled through the whole blood for fifteen to eighteen minutes at a rate which just permitted the bubbles to be counted and which did not give rise to too much frothing. The remaining steps in the technic were the same as those for untreated blood.

*Results*—Complete hemolysis occurred in sodium chloride concentrations between 0.25 and 0.32 per cent; partial hemolysis in concentrations between 0.33 and 0.45 per cent (Table 2). These findings are practically the same as those for untreated blood. There is, however, a tendency to a somewhat greater stability in aerated cells.

TABLE 2.—DEGREE OF HEMOLYSIS: AERATED BLOOD

| BLOOD SAMPLE | COMPLETE HEMOLYSIS | | PARTIAL HEMOLYSIS | | NO HEMOLYSIS | |
|---|---|---|---|---|---|---|
| | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests |
| 1 | 0.25–0.32 | 8 | 0.33–0.45 | 13 | 0.46–0.60 | 15 |
| 2 | 0.25–0.32 | 8 | 0.33–0.40 | 8 | 0.41–0.60 | 20 |
| 3 | 0.25–0.32 | 8 | 0.33–0.43 | 11 | 0.44–0.60 | 17 |
| 4 | 0.25–0.31 | 7 | 0.32–0.41 | 10 | 0.42–0.60 | 19 |

### 3. *Effect of Pure Carbon Monoxide on Fragility of Red Blood Cells*

*Procedure*—Carbon monoxide was generated by passing pure sulphuric acid (specific gravity 1.84) into pure formic acid. The gas was led from the generating flask through soda lime into the blood at the same rate as was used for ordinary aeration in experiment 2. The usual procedure was then followed.

*Results*—There was complete hemolysis in sodium chloride concentrations between 0.25 and 0.33 per cent; partial hemolysis in concentrations between 0.34 and 0.45 per cent (Table 3). The degree of hemolysis is just slightly greater than in untreated blood.

TABLE 3.—DEGREE OF HEMOLYSIS: BLOOD TREATED WITH CARBON MONOXIDE

| BLOOD SAMPLE | COMPLETE HEMOLYSIS | | PARTIAL HEMOLYSIS | | NO HEMOLYSIS | |
|---|---|---|---|---|---|---|
| | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests |
| 1 | 0.25–0.32 | 8 | 0.33–0.45 | 13 | 0.46–0.60 | 15 |
| 2 | 0.25–0.33 | 9 | 0.34–0.42 | 9 | 0.43–0.60 | 18 |
| 3 | 0.25–0.33 | 9 | 0.34–0.43 | 10 | 0.44–0.60 | 17 |

### 4. *Effect of Illuminating Gas on Fragility of Red Blood Cells*

*Procedure*—The gas passed from the jet through calcium chloride and then into whole blood at a rate approximating as nearly as possible that used for ordinary aeration. The usual procedure was then followed.

*Results*—Complete hemolysis occurred in concentrations ranging from 0.25 to 0.37 per cent; partial hemolysis in concentrations from 0.37 to 0.48 per cent (Table 4). The degree of hemolysis shows a marked increase over that produced by carbon monoxide.

TABLE 4.—DEGREE OF HEMOLYSIS: BLOOD TREATED WITH ILLUMINATING GAS

| BLOOD SAMPLE | COMPLETE HEMOLYSIS | | PARTIAL HEMOLYSIS | | NO HEMOLYSIS | |
|---|---|---|---|---|---|---|
| | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests |
| 1 | 0.25–0.36 | 12 | 0.37–0.45 | 9 | 0.46–0.60 | 15 |
| 2 | 0.25–0.36 | 12 | 0.37–0.48 | 12 | 0.49–0.60 | 12 |
| 3 | 0.25–0.37 | 13 | 0.38–0.48 | 11 | 0.49–0.60 | 12 |

### *5. Effect of Automobile Exhaust Gas on Fragility of Red Blood Cells*

*Procedure*—The exhaust gas was collected in special sampling flasks. It was then led through calcium chloride into the blood, at the rate used previously. Other steps in the procedure were the same as in the foregoing experiments.

*Results*—There was complete hemolysis in sodium chloride concentrations ranging from 0.25 to 0.36 per cent; partial hemolysis occurred in concentrations between 0.37 and 0.47 per cent (Table 5). The effect of automobile exhaust gas on the fragility of the red blood cells is, therefore, somewhat less than that of illuminating gas.

TABLE 5.—DEGREE OF HEMOLYSIS: BLOOD TREATED WITH AUTOMOBILE EXHAUST GAS

| BLOOD SAMPLE | COMPLETE HEMOLYSIS | | PARTIAL HEMOLYSIS | | NO HEMOLYSIS | |
|---|---|---|---|---|---|---|
| | % Concn. NaCl | No. of Tesst | % Concn. NaCl | No. of Tests | % Concn. NaCl | No. of Tests |
| 1 | 0.25–0.35 | 11 | 0.36–0.44 | 9 | 0.45–0.60 | 16 |
| 2 | 0.25–0.36 | 12 | 0.37–0.46 | 10 | 0.47–0.60 | 14 |
| 3 | 0.25–0.36 | 12 | 0.37–0.47 | 11 | 0.48–0.60 | 13 |

### *6. Effect of the Various Gases on Hydrogen Ion Concentration*

*Procedure*—Sodium chloride solution (0.9 per cent) was adjusted to a hydrogen ion concentration of 7.4. Samples of this solution were treated with air, carbon monoxide, illuminating gas, and automobile exhaust gas, in accordance with the procedure used for the blood. The hydrogen ion concentration was then determined. These determinations were repeated, using a buffer solution (M/15 $Na_2HPO_4$ $2H_2O$:M/15 $KH_2PO_4$ in the proportions 80.4:9.6), the hydrogen ion concentration of which was 7.35.

*Results*—All the sodium chloride samples (hydrogen ion concentration 7.4) lost their alkalinity very rapidly. With the buffer solution, the effect was not so marked. Carbon monoxide and illuminating gas lowered the hydrogen ion concentration to 7.3. The air treated sample and the exhaust gas sample had hydrogen ion concentrations of 7.2 and 7, respectively.

The hemolysis produced in experiments 1 to 5 cannot be due to any change in hydrogen ion concentration, since the buffering action of the blood is greater than that of the phosphate solution as shown in experiment 6.

## Summary

Experiments are reported which were undertaken for the purpose of studying the effect of pure carbon monoxide, illuminating gas, and automobile exhaust gas on the fragility of red blood cells. A comparison of the hemolysis observed under the different conditions of exposure is given in Table 6.

When normal blood was exposed to pure carbon monoxide gas under laboratory conditions, there was no increase in the fragility of the red blood cells.

Normal blood exposed to both illuminating gas and automobile exhaust gas, under the same laboratory conditions, showed a tendency to a somewhat increased hemolysis of the red cells. The increase in hemolysis due to automobile exhaust gas is slightly less than that produced by illuminating gas.

The hemolyzing effect of these gases would seem therefore to be due to the presence of other toxic constituents rather than to their carbon monoxide content.

Table 6.—Comparison of Hemolysis of Red Blood Cells under Different Conditions of Exposure

| Treatment | No. of tests | Percentage showing | | | Average point* of: | |
|---|---|---|---|---|---|---|
| | | Complete Hemolysis | Partial Hemolysis | No Hemolysis | Complete Hemolysis | Beginning Hemolysis |
| None | 108 | 24.1 | 28.7 | 47.2 | 0.33 | 0.43 |
| Aeration | 144 | 21.5 | 29.2 | 49.3 | 0.32 | 0.42 |
| Carbon monoxide | 108 | 24.1 | 29.6 | 46.3 | 0.33 | 0.43 |
| Illuminating gas | 108 | 34.3 | 29.6 | 36.1 | 0.36 | 0.47 |
| Automobile exhaust gas | 108 | 32.4 | 27.8 | 39.8 | 0.36 | 0.46 |

*Percentage concentration of sodium chloride.

The hemolysis produced by the several treatments outlined was not due to a change in the hydrogen ion concentration of the blood.

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# CARBON MONOXIDE HEADACHE

MAY R. MAYERS, M.D.

The typical carbon monoxide headache has been described many times and is familiar to all persons who have been exposed even to relatively small amounts of this gas. It is a pounding and intense headache—either basal or frontal—which is associated with nausea, and is increased by bending. Clinically, it strongly resembles headaches seen in persons suffering from brain tumor or other conditions characterized by increased intra-cranial pressure. Pathologically it has been shown to be due to edema of the brain, and this has been the rationale for the relief which follows the administration of magnesium sulphate. There are certain aspects of the headache, however, which have not been sufficiently considered, and which present problems the answer to which might be of fundamental importance in clarifying our understanding of the general toxicological effects of carbon monoxide gas.

It is the generally accepted view—particularly in this country—that carbon monoxide gas is not a tissue poison, and that all of its pathological effects may be fully explained on the basis of the anoxemia, or tissue asphyxia, which it produces. That its principal effects are due to this action is established beyond any question—particularly in acute poisoning. When only relatively small amounts of this gas are absorbed, however, the anoxemia theory does not seem to provide as complete an explanation for all of the manifestations of poisoning which one encounters. It does not seem, for example, to fully account for the typical carbon monoxide headache.

The most puzzling, and at the same time the most important, feature of the carbon monoxide headache is that it comes on when there is as little as 10 per cent saturation of the blood with carbon monoxide gas—or, at times, even less. Obviously, with so low a saturation, the oxygen reserves of the body are in no way compromised; there is still an ample oxygen supply to tissue cells, and there would seem to be no very good reason why an edema of the brain should develop so early.

If one follows this line of thought further, one is impressed with the extraordinary fact that in other conditions or diseases—where there is no exposure whatever to carbon monoxide gas—a far greater degree of anoxemia is not characteristically associated with

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this particular type of headache. In ascents in balloons, or in aeroplanes, for example; in severe anemias, or leukemias, where the hemoglobin may drop to 20 or 30 per cent; in pneumonia, where the anoxemia to tissue cells is sometimes so severe as to threaten life itself, and in other conditions which could be mentioned, headache, analogous to the carbon monoxide headache, is neither an early nor a prominent part of the symptom complex. Indeed, there may be no headache whatever. If there is a corresponding edema of the brain in these other conditions of oxygen deficiency, for some reason or other, its clinical manifestations are, apparently, not precisely the same as those in carbon monoxide poisoning.

## Headache Characteristic

On exposure even to very small amounts of carbon monoxide gas, the headache is a characteristic and early symptom. Clinically, it is always the same kind of a headache, and is so intense as to completely dominate the picture—putting every other discomfort into the background as far as the patient is concerned. Furthermore, while there are, normally, enormous differences in the susceptibility of individuals to toxic substances generally, these differences are not nearly so great in the matter of susceptibility to this headache. Practically everyone exposed to carbon monoxide gas experiences it.

In a previous article* the question of the relative toxicity of pure carbon monoxide, and carbon monoxide in mixed gases (such as the exhaust gas from automobiles and illuminating gas), was discussed. It was suggested that the greater toxicity of the mixed gases may, in part at least, account for those elements in the clinical pictures which have always been difficult to interpret solely in terms of oxygen deficiency (anoxemia). This may possibly be the explanation of the carbon monoxide headache.

In the absence of further evidence, it would seem as though one must face the fact that the anoxemia theory of carbon monoxide intoxication, as we now understand it, does not seem to explain fully the carbon monoxide headache—the fact that it develops regularly on exposure to carbon monoxide gas when tissue anoxemia is relatively slight, and may be absent in other conditions where there is a very severe anoxemia but no exposure to this gas. Most of the work that has been done has been with acute carbon monoxide poisoning where tissue asphyxia is intense. It is conceivable at

* M. R. Mayers: Exposure to Carbon Monoxide in Mixed Gases, *The Industrial Bulletin,* September 1937.

least that under these circumstances it would be most difficult to dissociate and evaluate any other relatively less important mechanisms which may be at work at the same time. Perhaps such mechanisms can only be detected by studying persons exposed to low concentrations of the gas where tissue anoxemia is never sufficiently great to be physiologically important.

It is believed that a better understanding of whatever factors are involved in the production of the carbon monoxide headache would contribute materially to our understanding of other systemic effects of carbon monoxide inhalation—particularly under conditions of industrial exposure where, generally speaking, only the relatively low concentrations of the gas prevail.

# EXPOSURE TO CARBON MONOXIDE GAS IN GARAGES

May R. Mayers, M.D.

In all, fifty garage workers were examined. Since it was the primary object of the study to determine the effects of prolonged exposure to relatively low concentrations of carbon monoxide gas, all examinations were made in the plants on regular work days. The series under consideration does not therefore include any cases of acute carbon monoxide poisoning.

*History*—In each case a careful history was taken, which in addition to the usual clinical history included inquiry as to the duration of employment in their present occupation; a history of previous occupations with a view to ascertaining previous exposure either to carbon monoxide gas or to any other of the industrial poisons in industry; the number of acute attacks of carbon monoxide poisoning sustained in the past, if any; and the condition of the patient's health, as nearly as it could be ascertained from the history, previous to any exposure to carbon monoxide gas.

*Scope of Examination*—A complete physical examination was made in each case. In addition to the usual examination of the heart and lungs, the pulse and respiratory rates were carefully noted. In each case these were counted for two minutes, so as to obtain greater accuracy. Blood pressure readings—both systolic and diastolic—were taken by means of a mercury sphygmomanometer. In some instances the pulse and blood pressure readings were taken at the beginning and end of the day, and these were compared.

In addition, routine blood counts were made in each case, including:

(1) Hemoglobin determination.
(2) Red blood-cell count.
(3) White blood-cell count.
(4) Morphological examination of the blood.

In the case of 27 men, further blood determinations were made as follows:

(1) Carbon monoxide content of the blood.
(2) Oxygen content.
(3) Oxygen capacity.
(4) Carbon dioxide content.
(5) Serum potassium.

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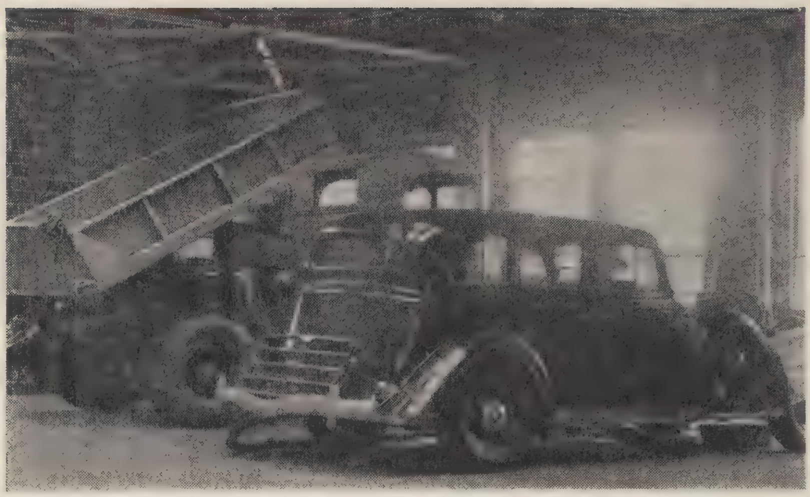
*Reprinted from N. Y. State Dept. Labor, Special Bulletin, Carbon Monoxide Poisoning in Industry, 1930.*



**Garage Ventilation**

*Air Tests*—In the case of each plant several tests were made for purposes of determining the carbon monoxide content of the air, samples being taken in various parts of the room. The carbon monoxide content of the air ranged from 2.3 parts per 10,000 in those parts of the rooms not immediately adjacent to cars giving off an exhaust, to 11 parts per 10,000 two feet back of cars which were exhausting gasoline fumes at the time the air samples were taken. The men were doing their regular work which necessitated their going about from one part of the workroom to another, so that they were exposed to varying concentrations of the gas, in the course of their day's work. They were in the habit of slipping out of doors for a few minutes now and again during the day to get some fresh air.

*Duration of Exposure*—The duration of their consecutive exposure to carbon monoxide either in the plant in which they were examined or in previous occupations ranged from 1.5 to 15 years. In all cases the men were exposed to the particular concentrations of carbon monoxide prevailing in their plant for from approximately two to four hours on the day the examinations were made, before they were examined, and blood tests taken.* All examinations were made in the rooms in which they worked, so that there was no period of waiting in an atmosphere free from carbon monoxide just previous to the examination.

* This duration of exposure on the day of examination is not accurate, since it was believed that some of the men went out of doors during this period.

## Symptomatology

Writers on carbon monoxide poisoning have emphasized the difference between acute poisoning due to sudden exposures to large concentrations of the gas, and chronic poisoning due to long-continued exposure to small amounts of carbon monoxide. In the former the severity of the symptoms will, within the limits of individual variation, be a direct measure of the degree of exposure and the concentration of carbon monoxide in the victim's blood. It will be proportional, generally speaking, to the product of its concentration in the atmosphere, multiplied by the length of time it was breathed. In chronic poisoning, on the other hand, no such simple proportion exists. The symptoms may be exaggerated in any given case, at a given time, due to the cumulative degenerative effects of former exposures. Conceivable, too, in other cases, they may not be as severe as might be expected on the basis of the carbon monoxide content of the blood; due to some compensating mechanisms which the body has been able, after repeated exposures, to call into play. Certain individuals may acquire tolerance after prolonged and repeated exposures, while the compensatory mechanism may never function properly in other cases; or, after being called upon too often, it may gradually fail to do the work. The increase of red-cells as a compensatory mechanism, which in some cases has been followed by the gradual onset of severe anemia, if the exposure continues for a sufficiently long time, has already been discussed.

It is of interest to note that the subjective symptoms complained of were those ordinarily classified as "prodromal" (warning)

TABLE I. SYMPTOMS OF WHICH WORKERS COMPLAINED

| Symptoms | Number of cases | Percentage of total cases |
|---|---|---|
| Headache | 37 | 74 |
| Dizziness | 25 | 50 |
| Burning of the eyes | 16 | 32 |
| Gastro-intestinal disturbances | 12 | 24 |
| Drowsiness | 14 | 28 |
| Faintness | 6 | 12 |
| Insomnia | 10 | 20 |
| Cardiac disturbances | 9 | 18 |
| Mental depression | 6 | 12 |
| Irritability | 15 | 30 |
| Ringing in the ears | 5 | 10 |
| Cough | 14 | 28 |
| No symptoms | 4 | 8 |

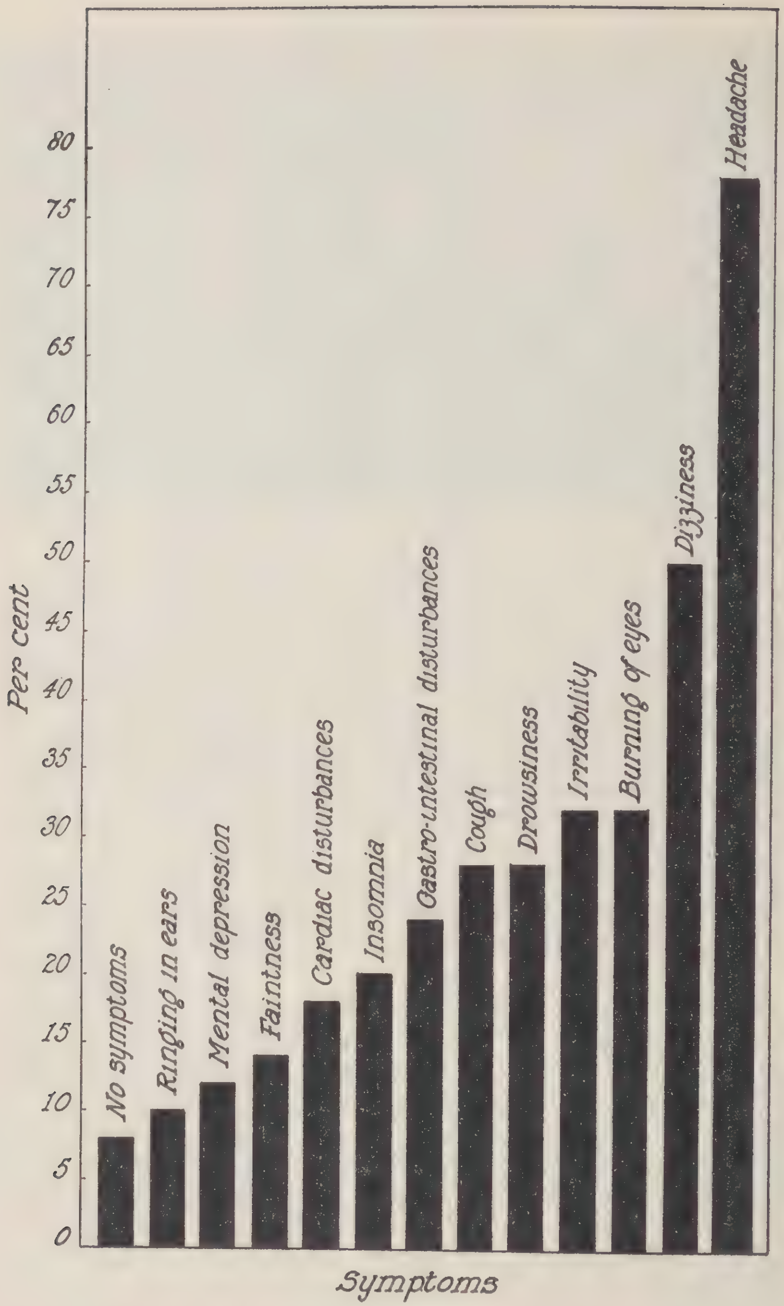



Chart 1. Symptoms

symptoms of acute carbon monoxide asphyxia, and yet in the case of these men these symptoms were not prodromata to be followed by an acute attack of carbon monoxide asphyxia. They were merely the chronic symptoms which troubled them intermittently while at work, and frequently after they went home for the night.

The symptoms listed were not all experienced by the workers on the day that the examinations were made, but represented their experience during a period of weeks or months of exposure to carbon monoxide gas in the same plant. This explains in part, at least the lack of correlation found between symptoms and the carbon monoxide content of the blood at the time of the examination. The symptoms are listed in Table I.

It will be observed that the majority of the symptoms are referable to the nervous system, and that as is to be expected the carbon monoxide headache predominates. Only four workers claimed to experience no symptoms whatever, and no discomfort as a result of their exposure.

*Headache*—Of all the nervous manifestations resulting from exposure to carbon monoxide, perhaps the most striking is the headache, which is peculiarly intense and of long duration. This headache is in itself responsible for more loss of time on the part of workers exposed to relatively low concentrations of carbon monoxide gas than any one or group of symptoms complained of. While other symptoms such as dizziness, smarting of the eyes, nausea, drowsiness, and lack of muscular coordination are all responsible for impaired efficiency, and undoubtedly predisposed to accidents, the carbon monoxide headache is the usual and chief cause for actual loss of time.

The headache, which was absent in only 13 cases, was described as extremely intense, characterized by pounding at the temples or at the back of the head. It was increased on bending; was occasionally associated with nausea, and was frequently of many hours duration. It was also found to be associated with marked pallor, and increased nervous irritability. This pallor, which will be discussed in detail later (see p. 27) was of an ashen hue and was strikingly suggestive of the ashen pallor seen among lead workers. It was extreme in the men who were suffering from headache, even in the cases where there was initial flushing of the skin previous to its onset. The association of pallor with a basal headache which was increased by bending and frequently accompanied by nausea, suggested very strongly that the headache might be due to increased intra-cranial pressure. Indeed, its similarity clinically to the headache associated with increased intra-cranial pressure due

to brain tremor was rather impressive. It has already been shown that there is a pathological basis for this clinical observation. Klebs and Lewin both reported hyperaemia and dilatation of the large vessels of the brain. Furthermore, various investigators have shown that there is actually an increased intra-cranial pressure in these cases.

One of the striking features of the carbon monoxide headache is not only its duration, but the fact that its onset may be delayed. and frequently is delayed, until an hour or even several hours after the individual has left the workshop. In other words, it occurs after there is every reason to believe that whatever carbon monoxide was present in the blood stream has been entirely eliminated. This fact always causes considerable surprise.

Although difficult of interpretation for a time, it has been demonstrated by trephine experiments upon experimental animals that during the period of recovery after exposure to carbon monoxide gas there is an increase in intra-cranial pressure which is far greater than any which may have been present during the actual exposure. This would seem to account for the fact that the headache may begin during the period following exposure to the gas. Frequently workers complain that they have no headache until they reach home in the evening. It may then last for a number of hours. frequently for as many as ten hours, and in some cases even longer. It is so intense as to interfere with sleep and is not ordinarily relieved by any medication.*

*Relation of Symptoms to Hemoglobin Content of the Blood*—Because the hemoglobin content of the blood was regarded as in a measure indicative of the ability of the worker to compensate for the oxygen deficiency of his blood and tissues entailed by exposure to carbon monoxide gas, those symptoms complained of were correlated with the hemoglobin content of the blood, as shown in Table II.

While on the whole, workers with a hemoglobin of 80% or less appeared to suffer greater discomfort than did the others, this was by no means invariably the case. Indeed, even those with a very high hemoglobin content of the blood and a high red cell count appeared to suffer from a number of symptoms, particularly from headache. Theoretically, from the standpoint of exposure to *pure* carbon monoxide gas, the worker whose blood has an unusually high hemoglobin content and an increased number of red blood

* The use of hypertonic salt solutions to reduce the increased intracranial pressure has been suggested. Large doses of Epsom salts have been advocated for this purpose. They have not been experimented with, however, by the present investigation to date.

Table II. Showing Relation of Hemoglobin Content of Blood to Symptoms

| Hemoglobin, Per Cent | Symptoms | | | Total number of cases |
|---|---|---|---|---|
| | Headache | Other | None | |
| Under 60 | 1 | 1 | ....... | 1 |
| 60–70 | 5 | 4 | ....... | 5 |
| 71–80 | 6 | 5 | ....... | 6 |
| 81–90 | 6 | 6 | 1 | 8 |
| 91–100 | 6 | 7 | 1 | 7 |
| 101–110 | 5 | 10 | ....... | 10 |
| 111–120 | 5 | 4 | 1 | 6 |
| 121–130 | 2 | 1 | 1 | 3 |
| 131–140 | ....... | 1 | ....... | 1 |
| 141–150 | 2 | 2 | ....... | 2 |
| 151–160 | 1 | ....... | ....... | 1 |

cells is undoubtedly in a better position than his fellows to keep his tissues supplied with a quantity of oxygen more nearly approaching the normal and should therefore be expected to be less subject to discomfort when exposed to the gas. In the case of exposure to exhaust gas, however, it appears that severe symptoms occur even in those whose blood-forming organs seem to be compensating fairly well. This suggests the possibility therefore that some of the contaminants which are present in the gas may be responsible—at least in part. Undoubtedly, they play an important part in altering the symptom complexes of those exposed to the gas, and complicate the clinical picture considerably.

## Physical Findings

*Pallor*—On superficial inspection of a considerable number of workers exposed to carbon monoxide gas in one form or another—particularly those exposed to illuminating gas and the exhaust gas from automobiles—one was impressed with the existence of a marked pallor, very suggestive of the pallor seen among lead workers. In the case of the 50 garage workers examined, special note was made as to a possible correlation between this pallor and the hemoglobin content of the blood, particularly in view of the fact that exposure to pure carbon monoxide tends to cause a polycythemia, i.e., a marked increase in the hemoglobin and the red cell count, rather than an anemia. Marked pallor was noted in 38 cases, or 76%. In 26 cases pallor was observed where the hemoglobin was 80% or higher. Thirteen were found in men having a hemoglobin over 100%. In 25 cases the hemoglobin was 100% or less.

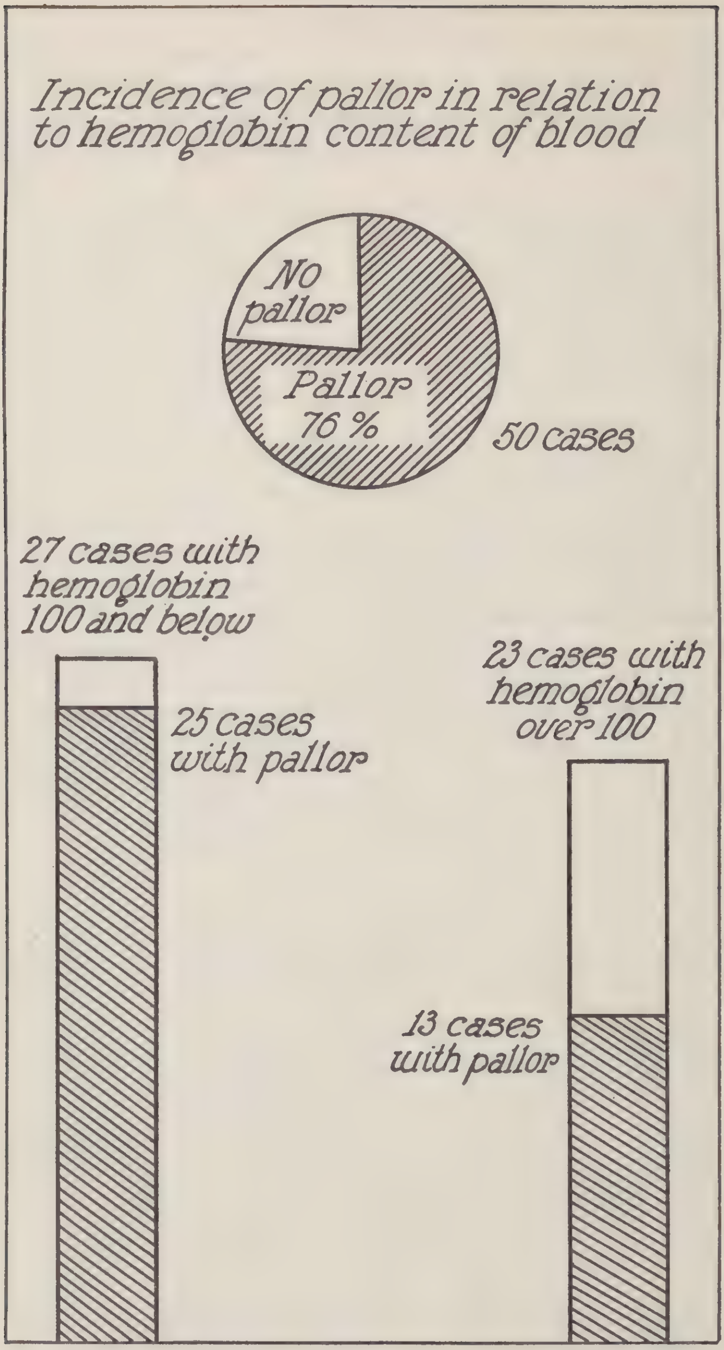



**Chart 2. Incidence of Pallor in Relation to Hemoglobin Content of Blood**

Apparently, pallor may be present even where there is a high hemoglobin content of the blood. This may perhaps be due to a vaso-constriction of the superficial capillary circulation, somewhat analogous to that found in persons exposed to lead. Its association with headache has already been commented upon.

*The Cardio-Vascular System*—Routine examination of the cardio-vascular system by the ordinary clinical methods did not reveal any abnormality worthy of comment, with the exception of the fact that the pulse rate was almost uniformally higher than normal. See Chart 3.

In approximately one-third of the cases, the pulse rates ranged from 91–100 beats per minute. In 68% of the cases, it was above 90. The average for the series was 101.84 as compared with the normal 72. The significance of a rapid pulse rate which is maintained over a long period of time will be touched upon briefly in the discussion of the increased respiratory rate which follows.

Cardiac symptoms were complained of by 9 of the workers examined. These are shown in Table III.

TABLE III. CARDIAC SYMPTOMS

| SYMPTOM | Number of cases |
|---|---|
| Palpitation on exertion | 5 |
| Pre-cordial pain | 2 |
| Shortness of breath | 2 |

Several of these men stated that the cardiac symptoms of which they complained always followed exposure to the higher concentrations of the gas—as, for example, when they were lying underneath a car making repairs, while the engine was running.

Clinical examination of the heart in these men revealed no definite abnormality; and even functional tests of the simpler sort brought out very little of significance. It would be of great interest to determine whether, if these workers were examined by means of the electro-cardiograph, anything could be found to account for their symptoms. It is a well-known fact for example, that in the earliest stages of myocarditis, as well as in disease of the coronary arteries (precursor to angina pectoris) which supply nutrition to the heart muscle, clinical examination of the patient may reveal

## Table IV. A Clinical Examination of Fifty Garage Workers

| Case No. | Age | Duration of employment | Symptoms Complained of by Workers: Cough | Headache | Dizziness | Irritability | Mental depression | Faintness | Drowsiness | Insomnia | Burning of eyes | Ringing in ears | Gastro-intestinal disturbances | Cardiac disturbances | Physical Examination: Pulse | Respiration | Pallor | Tremor | Reflexes* | Blood Pressure: Systolic | Blood Pressure: Diastolic | Blood Count: Hemoglobin, per cent | Red blood cells (in thousands) | White blood cells | Comments |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 30 | 12 yrs. | ..... | Rare. | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 100 | 24 | ++ | ..... | ++ | 145 | 80 | 75 | 5,472 | 6,200 | |
| 2 | 32 | 4 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | ..... | ..... | 75 | 20 | + | ..... | + | 90 | 60 | 105 | 3,920 | 6,500 | "Head never clear." |
| 3 | 27 | 7 yrs. | ..... | ..... | + | + | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | 80 | 30 | ..... | ..... | + | 110 | 70 | 107 | 5,088 | 7,100 | "Hard to get up in morning because of dizziness. |
| 4 | 39 | 4 yrs. | ..... | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 96 | 20 | ++ | ..... | ++ | 112 | 70 | 67 | 4,736 | 9,200 | |
| 5 | 41 | 4 yrs. | + | + | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | Palpitation + | 120 | 26 | + | + | + | 160 | 90 | 101 | 5,072 | 6,850 | |
| 6 | 23 | 4 yrs. | ..... | ++ | + | ++ | ..... | ..... | ..... | ..... | ..... | ..... | Poor appetite | ..... | 92 | 24 | + | + | ++ | 150 | 50 | 83 | 4,416 | 11,200 | Poor muscular coordination. |
| 7 | [illegible] | 14 yrs. | ..... | ++ | + | + | ..... | + | + | + | + | ..... | ..... | Crecordial pain | 130 | 28 | ++ | ..... | + | 90 | 60 | 40 | 4,208 | 6,150 | "Always tired." |
| 8 | 23 | 5 yrs. | ..... | + | + | ..... | ..... | ..... | + | + | ..... | ..... | Nausea | ..... | 120 | 24 | + | ..... | + | 120 | 45 | 84 | 5,984 | 7,450 | |
| 9 | 35 | 4 yrs. | + | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 82 | 20 | ..... | + | + | 100 | 65 | 146 | 4,944 | 10,850 | |
| 10 | 27 | 3 yrs. | ..... | ++ | + | ..... | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | 93 | 24 | + | + | ++ | 98 | 60 | 109 | 4,432 | 7,500 | |
| 11 | 27 | 2 yrs. | + | ++ | + | + | ..... | ..... | + | ..... | ..... | ..... | ..... | ..... | 92 | 24 | + | + | + | 95 | 62 | 102 | 3,944 | 10,000 | "Always tired in the morning." |
| 12 | 26 | 2 yrs. | ..... | ++ | + | + | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | 98 | 30 | ++ | ..... | ++ | 105 | 75 | 60 | 4,272 | 8,220 | |
| 13 | 51 | 25 yrs. | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 84 | 22 | + | + | ++ | 155 | 105 | 96 | 4,224 | 6,150 | |
| 14 | 18 | 4 yrs. | ..... | + | + | ..... | ..... | ..... | + | ..... | ..... | ..... | ..... | Palpitation + | 120 | 34 | + | ..... | + | 120 | 55 | 112 | 4,688 | 6,200 | Overcome 2 years ago. |
| 15 | 38 | 4 yrs. | ..... | ++ | + | + | ..... | ..... | ..... | ..... | + | + | Nausea | ..... | 100 | 30 | + | ++ | ++ | 95 | 58 | 96 | 4,896 | 6,100 | |
| 16 | 19 | 2 yrs. | + | ++ | + | ..... | ..... | ..... | ..... | ..... | + | ..... | Nausea | ..... | 110 | 28 | + | ++ | ++ | 110 | 48 | 114 | 4,656 | 5,350 | |
| 17 | 26 | 3 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | + | ..... | + | ..... | ..... | ..... | 100 | 28 | + | ..... | + | 115 | 65 | 134 | 4,832 | 7,650 | |
| 18 | 18 | 4 yrs. | ..... | ++ | + | + | ..... | + | ..... | ..... | ..... | + | ..... | ..... | 130 | 30 | + | + | ++ | 80 | 55 | 79 | 4,224 | 7,400 | "Weak and shaky in knees." |
| 19 | 25 | 9 yrs. | + | ++ | + | + | + | ..... | ..... | ..... | + | ..... | ..... | Dyspuvea on exertion | 122 | 26 | ++ | + | ++ | 138 | 80 | 60 | 4,557 | 8,100 | Feels worse after smoking. |
| 20 | 38 | 10 yrs. | + | + | ..... | ..... | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | 100 | 28 | ..... | ..... | + | 130 | 65 | 101 | 4,488 | 9,600 | "Throbbing in head on bending." |
| 21 | 29 | 4 yrs. | ..... | ..... | + | + | ..... | + | ..... | ..... | ..... | ..... | Nausea | ..... | 114 | 24 | ++ | + | ++ | 90 | 55 | 61 | 4,960 | 8,900 | |
| 22 | 25 | 10 yrs. | ..... | Rarely | ..... | ++ | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 110 | 22 | ++ | + | ++ | 160 | 90 | 77 | 5,264 | 6,250 | |
| 23 | 36 | 2 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 100 | 24 | ..... | ..... | + | 125 | 68 | 119 | 4,784 | 6,400 | |
| 24 | 30 | 6 yrs. | ..... | ++ | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 90 | 24 | ..... | + | + | 110 | 80 | 154 | 5,872 | 9,300 | |
| 25 | 18 | 2 yrs. | + | ++ | + | + | ..... | ..... | + | ..... | + | ..... | Pain in pit of stomach | ..... | 124 | 30 | ++ | + | + | 122 | 58 | 65 | 4,808 | 11,000 | "Always tired and sleepy." |
| 26 | 41 | 10 yrs. | + | + | ..... | ..... | + | ..... | + | + | ..... | ..... | ..... | ..... | 110 | 26 | + | ..... | + | 118 | 70 | 86 | 4,120 | 7,400 | |
| 27 | 27 | 5 yrs. | ..... | + | + | ..... | ..... | ..... | + | ..... | ..... | ..... | ..... | ..... | 120 | 28 | ..... | + | + | 110 | 64 | 117 | 5,200 | 5,150 | |
| 28 | 38 | 3 yrs. | ..... | + | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 100 | 22 | ..... | ..... | + | 120 | 72 | 104 | 4,980 | 7,000 | |
| 29 | 26 | 5 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 100 | 24 | ..... | ..... | + | 115 | 78 | 100 | 5,100 | 4,950 | |
| 30 | 34 | 2 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 90 | 24 | ..... | ..... | + | 135 | 62 | 89 | 4,000 | 5,220 | |
| 31 | 23 | 3 yrs. | + | + | ..... | ..... | ..... | ..... | ..... | ..... | + | ..... | ..... | ..... | 88 | 30 | + | ..... | + | 100 | 58 | 97 | 5,250 | 6,200 | |
| 32 | 19 | 1 yrs. | ..... | ++ | + | + | ..... | + | ..... | + | ..... | + | Nausea | Cardiac palpitation | 118 | 30 | ++ | + | ++ | 80 | 56 | 75 | 3,900 | 5,800 | |
| 33 | 56 | 20 yrs. | + | ++ | ..... | ..... | ..... | + | ..... | ..... | + | ..... | ..... | Palpitation. | 100 | 24 | ++ | + | ++ | 160 | 95 | 100 | 5,200 | 6,000 | "Get short of breath more often than before." |
| 34 | 42 | 8 yrs. | + | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 84 | 28 | + | ..... | + | 156 | 98 | 95 | 4,820 | 7,000 | |
| 35 | 29 | 1 yrs. | ..... | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 86 | 24 | + | ..... | + | 115 | 68 | 98 | 4,650 | 5,800 | |
| 36 | 46 | 8 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 80 | 22 | ..... | ..... | + | 130 | 70 | 124 | 5,250 | 6,600 | |
| 37 | 34 | 4 yrs. | ..... | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 78 | 24 | ..... | ..... | + | 125 | 72 | 130 | 6,100 | 6,500 | |
| 38 | 25 | 3 yrs. | ..... | ..... | ..... | ..... | ..... | ..... | + | + | ..... | ..... | Poor appetite | ..... | 84 | 28 | + | ..... | + | 110 | 78 | 105 | 5,200 | 6,100 | |
| 39 | 19 | 2 yrs. | ..... | ++ | ..... | + | + | ..... | + | + | + | ..... | Nausea | ..... | 120 | 30 | + | + | ++ | 130 | 60 | 78 | 4,300 | 5,800 | "Tire easily." |
| 40 | 50 | 15 yrs. | + | ..... | + | ..... | ..... | + | ..... | ..... | ..... | + | ..... | Palpitation. | 110 | 26 | + | ..... | + | 115 | 70 | 102 | 4,890 | 5,200 | "Dizzy when I bend over. |
| 41 | 43 | 11 yrs. | + | ++ | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 100 | 24 | + | ..... | + | 165 | 92 | 125 | 6,250 | 7,400 | |
| 42 | 26 | 7 yrs. | ..... | + | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | 90 | 24 | ..... | ..... | + | 115 | 78 | 120 | 6,500 | 8,200 | |
| 43 | 22 | 3 yrs. | ..... | ++ | + | ..... | ..... | ..... | + | ..... | ..... | ..... | Nausea | ..... | 112 | 28 | + | + | + | 94 | 52 | 88 | 4,250 | 9,000 | |
| 44 | 31 | 6 yrs. | ..... | ++ | + | + | + | ..... | ..... | + | ..... | ..... | Nausea | ..... | 80 | 22 | + | ..... | ++ | 89 | 65 | 85 | 5,250 | 4,900 | |
| 45 | 37 | 6 yrs. | ..... | + | ..... | + | ..... | ..... | + | + | ..... | ..... | ..... | Precordial pain | 96 | 28 | + | ..... | + | 104 | 80 | 108 | 5,890 | 5,250 | |
| 46 | 40 | 10 yrs. | ..... | + | + | ..... | ..... | ..... | ..... | ..... | + | ..... | ..... | Short of breath | 92 | 26 | + | + | ++ | 120 | 75 | 142 | 5,980 | 10,100 | |
| 47 | 33 | 8 yrs. | ..... | ++ | ..... | ..... | ..... | ..... | ..... | ..... | ..... | ..... | Poor appetite | ..... | 86 | 24 | + | ..... | + | 105 | 60 | 120 | 6,000 | 4,900 | |
| 48 | 20 | 1 yrs. | ..... | + | + | ..... | ..... | ..... | + | ..... | ..... | ..... | ..... | ..... | 88 | 28 | + | + | ++ | 98 | 56 | 79 | 4,200 | 8,400 | "Can't get any rest." |
| 49 | 27 | 3 yrs. | ..... | ++ | + | + | + | + | ..... | + | + | ..... | ..... | ..... | 104 | 32 | + | + | ++ | 120 | 78 | 90 | 4,900 | 9,000 | |
| 50 | 24 | 5 yrs. | ..... | ++ | + | ..... | ..... | ..... | ..... | + | + | + | ..... | ..... | 94 | 24 | + | + | ++ | 100 | 80 | 100 | 5,850 | 6,100 | |
| | | | 14 | 37 | 25 | 15 | 6 | 6 | 14 | 10 | 16 | 5 | 12 | 9 | Averag 101.84 | Average 25.88 | 38 | 23 | 50 | Average 117.08 | Average 69.12 | Average 98.04 | Average 4,915 | Average 7,191.8 | |

*Reflexes marked + are normal; those marked ++ are hyperactive. The knee jerks were the only reflexes tested.

nothing. Even between acute attacks of angina pectoris it is frequently impossible, without the assistance of the electro-cardiograph, to detect that anything is wrong. It was rather to be regretted, therefore, that the precise cardio-vascular status, particularly of those men complaining of cardiac symptoms, could not be investigated by means of the electro-cardiograph and a more definite conclusion arrived at. Such an investigation would be of supreme importance because of the cardiac sequelae known to result from exposure to high concentrations of carbon monoxide gas.

The blood pressure findings were also essentially within the range of normal, a few being high, and a few abnormally low, but the

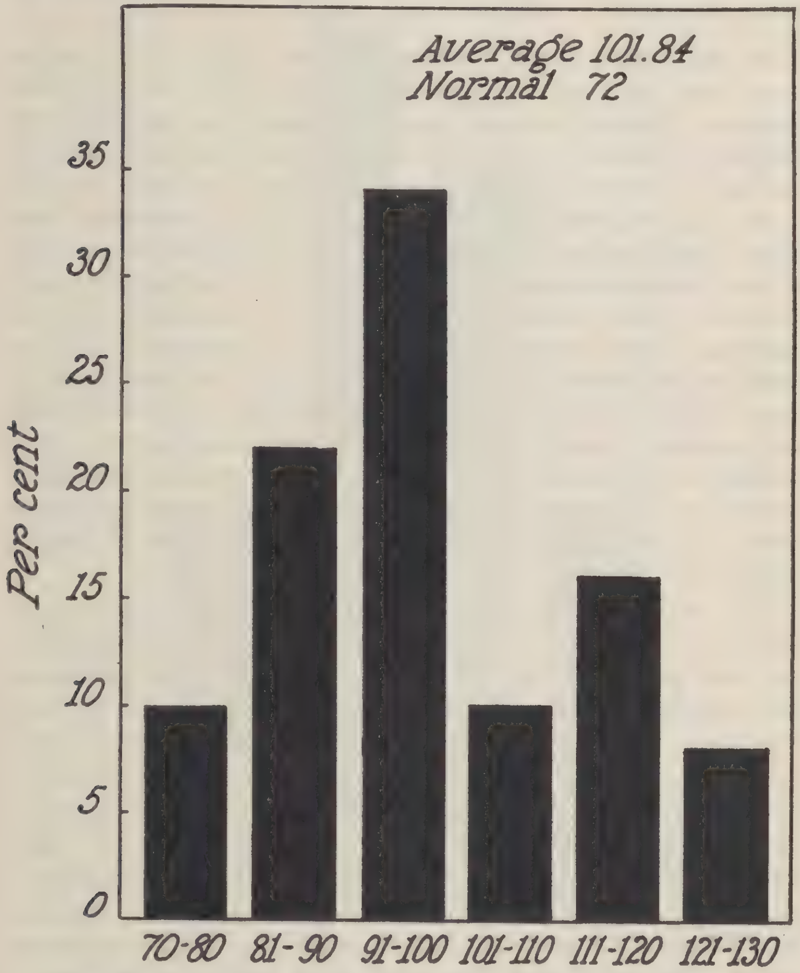



**Chart 3. Showing the Incidence (Per Cent) of Various Pulse Rates in 50 Garage Workers**

blood pressure readings showed a tendency, on the part of some of the workers, to drop considerably in the course of the day's work. In the case of others, blood pressure readings at the beginning and at the end of the day showed a distinct increase. There was apparently considerable individual variation in the reaction of the heart to the abnormal environment created not merely by the oxygen deficiency of the tissues due to absorption of carbon monoxide gas, and the maintenance of an abnormally high pulse and respiratory rate, but by the presence of the various toxic contaminants in exhaust gas. There appeared to be no correlation between cardiac symptoms and either the carbon monoxide or hemoglobin contents of the blood.

*The Respiratory System*—Despite the fact that 14 of the workers complained of cough, examination of the lungs was essentially negative. Because in many instances cough was associated with burning of the eyes, in the same individuals, there was some question as to whether both these symptoms were not the result of increased susceptibility to the gasoline fumes of an asthmatic character, rather than due to exposure to carbon monoxide per se. Some of these workers seemed to feel that they were particularly sensitive to the odor of exhaust gas as well, and a few believed they had "asthma." The precise relation to carbon monoxide absorption could not be determined one way or the other with any degree of security. The thought suggests itself, however, that workers who are peculiarly sensitive in this way, and who tend to cough when exposed to exhaust gas, might be more ready victims of pneumonia, should they be overcome by the gas, than those whose respiratory tracts are less sensitive. This is important enough to warrant further investigation.

The respiratory rate just as the pulse rate appeared to be rather high on the whole. Forty-two of the men examined had a respiratory rate over the normal 22. Twenty-four of the men had respiratory rates ranging from 26 to 33 per minute. The average for the series was 25.88. It might be well to call attention again to the fact that an environment which necessitates the continued maintenance of increased cardiac activity and increased respiration cannot be regarded as without potentialities for cardiac strain. It would seem a proper precaution that no worker be exposed to such an environment whose heart is not organically sound to begin with. While an individual with a perfectly normal heart may without doubt succeed in compensating perfectly well for the added strain which is thus put upon it, and may be entirely uninjured by it, an indi-

vidual whose heart is already either functionally or organically impaired would not ordinarily withstand the additional burden without the possibility of further injury.

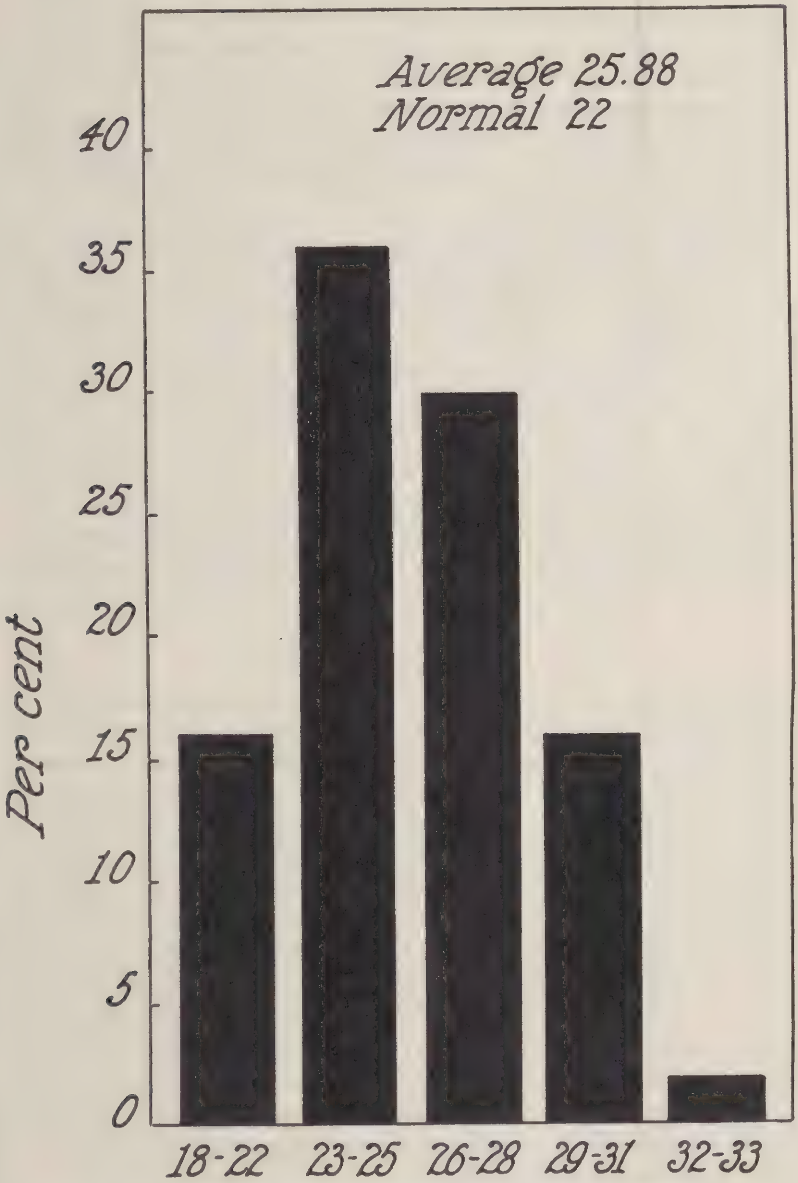



**Chart 4. Showing the Incidence (Per Cent) of Various Respiratory Rates in 50 Garage Workers**

*The Nervous System*—Examination of the nervous system was confined to an investigation of the existence of tremor, either of the hands or of the tongue; to the activity of the reflexes as measured by knee jerk, and to the general emotional and nervous stability. The symptoms referable to the nervous system have already been discussed. Tremor was found in 46% of the cases, and hyper-active knee jerks in 40%. Thirty per cent showed both tremor and hyper-active knee jerks. The fact that tremor might be due to many other causes, the principal one being chronic alcoholism, was borne in mind and those believed to be chronically addicted to alcohol were excluded from the series.

Workers exposed to exhaust gas appeared to be rather more suggestible on the whole than other workers who have been examined by the Bureau. It was further observed that they showed a greater emotional instability, on the whole, when faced with unfamiliar situations than have been observed in previous investigations. The laboratory apparatus which was brought into the plant, for example, caused far more uneasiness than had been met with previously, and this appeared to be particularly true where the carbon monoxide content of the air happened to be high. There was also to be observed a distinct increase in nervous irritability in those exposed to somewhat higher concentrations of the gas, such as following the making of repairs under a car.

## Laboratory Tests

In 50 cases, red and white blood-cell counts were made by the finger-prick method and in the usual manner, and smears were made for a differential count, and an examination of the morphology of the cells. In making the hemoglobin determinations, the Palmer colorimetric method was used. It was considered that the Sahli method would be inaccurate in these cases, because of the abnormal red color given to the blood by the carbon monoxide hemoglobin. The Palmer method is in principle dependent upon the formation of carbon monoxide hemoglobin and the color standards are prepared especially with reference to this. The Palmer method, therefore, automatically tends to eliminate any error which might occur due to the fact that part of the hemoglobin was combined with carbon monoxide and is abnormally red in color to begin with. The results were then expressed in percentages, according to the Newcomer Scale (16.92 grams of hemoglobin per 100 c. c. of blood representing 100 per cent).

In 27 cases the following additional blood determinations were made.

(1) Carbon monoxide content of the blood.
(2) Oxygen content of the blood.
(3) Oxygen capacity.
(4) Oxygen unsaturation.
(5) Carbon dioxide content of the blood.

In 11 cases the potassium content of the blood serum was also examined. The individual figures for all of these determinations may be seen by reference to Table V.

Blood samples were taken from the vein under albolene, with potassium oxalate as the anti-coagulant, and these were examined in the laboratory on the same day for oxygen content, hemoglobin and carbon dioxide content. Other blood samples were taken from the vein using no anti-coagulant. These were examined for serum potassium. The carbon monoxide content of the blood was made by the modified Van Slyke method.

TABLE V. BLOOD FINDINGS IN GARAGE WORKERS

| CASE NO. | Hemoglobin, per cent | Carbon monoxide content, per cent | Oxygen | | | Serum potassium | Carbon dioxide volumes, per cent |
|---|---|---|---|---|---|---|---|
| | | | (1) Content volumes, per cent | (2) Capacity volumes, per cent | (2)–(1) Unsaturation volumes, per cent | | |
| 1 | 75 | 14.3 | 11.3 | 14.9 | 3.6 | 25.0 | 45.96 |
| 2 | 105 | 2.3 | 3.9 | 19.4 | 15.5 | 21.1 | 60.7 |
| 3 | 107 | ...... | 9.0 | 19.7 | 10.7 | 24.1 | 48.5 |
| 4 | 67 | 5.5 | 1.9 | 15.2 | 13.3 | 28.0 | 44.96 |
| 5 | 101 | 6.0 | 6.1 | 18.8 | 12.7 | 23.2 | 55.9 |
| 6 | 83 | 2.1 | 8.7 | 15.4 | 6.7 | 24.4 | 47.1 |
| 7 | 40 | 0.1 | 2.7 | 7.4 | 4.7 | 23.1 | 57.4 |
| 8 | 84 | 8.3 | 6.9 | 15.7 | 8.8 | 25.1 | 53.2 |
| 9 | 146 | 0.1 | 9.0 | 27.2 | 18.2 | ...... | 54.4 |
| 10 | 109 | 10.0 | 6.0 | 20.2 | 14.2 | ...... | 54.7 |
| 11 | 102 | 5.2 | 8.4 | 19.0 | 10.6 | ...... | 45.6 |
| 12 | 60 | 5.2 | 3.0 | 13.6 | 10.6 | 21.2 | 60.6 |
| 13 | 96 | 2.8 | 8.0 | 14.9 | 6.9 | ...... | 49.4 |
| 14 | 112 | 5.8 | 6.2 | 20.7 | 14.5 | ...... | 62.2 |
| 15 | 96 | 0.0 | 5.5 | 17.7 | 12.2 | ...... | 57.4 |
| 16 | 114 | 19.7 | 10.8 | 21.2 | 10.4 | ...... | 53.4 |
| 17 | 134 | 0.0 | 5.3 | 24.8 | 19.5 | ...... | 52.4 |
| 18 | 79 | 2.3 | 2.4 | 14.7 | 12.3 | ...... | 59.7 |
| 19 | 60 | 0.0 | 3.4 | 11.2 | 7.8 | ...... | 61.8 |
| 20 | 101 | 30.1 | 6.2 | 18.7 | 12.5 | ...... | 52.0 |
| 21 | 61 | 2.9 | 7.8 | 13.2 | 5.4 | ...... | 48.8 |
| 22 | 77 | 7.2 | 5.0 | 15.4 | 10.4 | ...... | 52.3 |
| 23 | 119 | 8.2 | 3.8 | 22.0 | 18.2 | ...... | 50.7 |
| 24 | 154 | 21.1 | 3.6 | 28.7 | 25.1 | ...... | 54.7 |
| 25 | 65 | 17.4 | 12.1 | 12.0 | ...... | ...... | 35.2 |
| 26 | 86 | 5.2 | 4.3 | 11.0 | 6.7 | ...... | 51.8 |
| 27 | 117 | 11.8 | 6.0 | 21.6 | 15.6 | ...... | 51.9 |

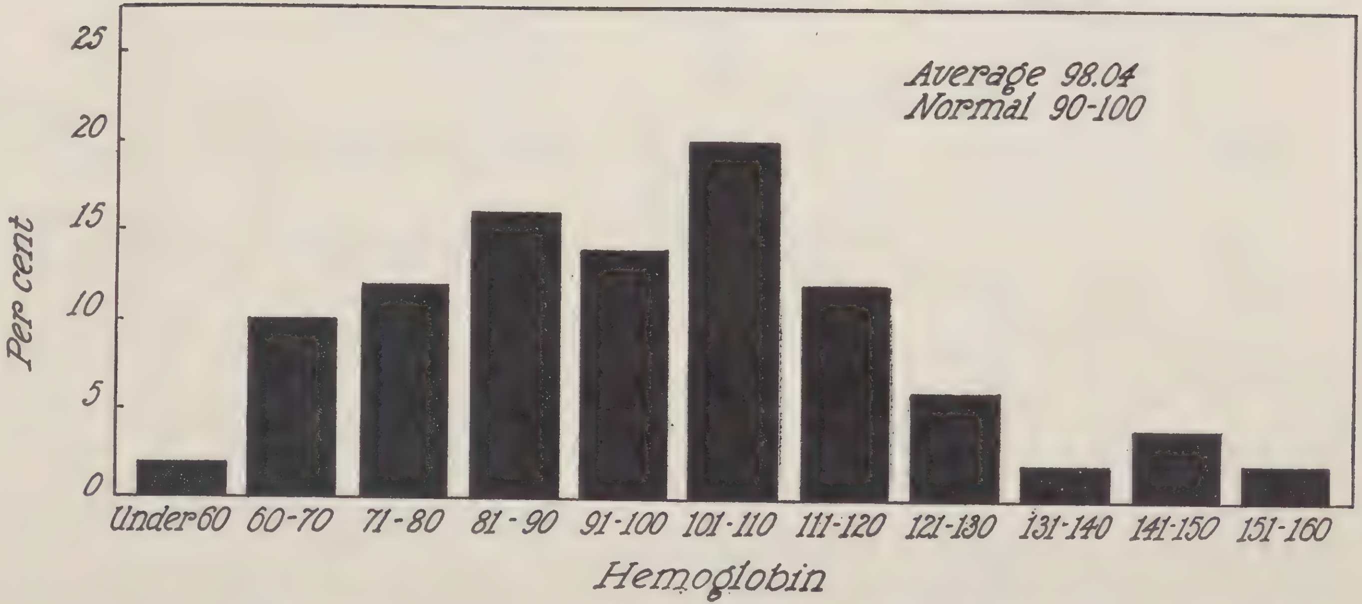



Chart 5. Showing the Incidence (Per Cent) of Various Amounts of Hemoglobin in the Blood of 50 Garage Workers

*Hemoglobin*—It will be remembered that when exposed to pure carbon monoxide, there is a tendency toward a polycythemia—i. e., an increase in the red cell count and the hemoglobin content of the blood. In the present series the hemoglobins range from 40% to 154%. It is rather interesting to note that the men were approximately evenly divided as between those having a hemoglobin of 100% and below, and those having a hemoglobin above 100%. Chart 5 shows that 54% had a hemoglobin of 100% or below, while in the case of 46% the hemoglobin was over 100%. The average hemoglobin was found to be 98.4%, as compared with the normal 90–100%. In workers exposed to exhaust gas, it has already been pointed out that the contaminants tend to complicate the blood picture. It has been a matter of general observation that many of these workers do not appear to compensate normally and show a tendency to an anemia. While this tendency may be the result of inability on the part of the blood-forming mechanism to compensate normally under conditions of exposure to carbon monoxide gas, the contaminants which enter into the picture must be taken into account. As mentioned before, there appeared to be no correlation between the hemoglobin content of the blood and either pallor or the symptoms complained of by the workers.

*Blood Count*—While on the whole there was a slight tendency to an increase in the red cell count, it was not found to be as high as might be expected. The same considerations already discussed in connection with the hemoglobin findings with reference to the effect of contaminants in altering the blood are equally applicable here.

The white cell count was essentially normal in all cases, as was also the differential count.

The morphology of the cells showed a marked difference from that found in lead workers. Even in the cases where there was considerable anemia—and it will be observed from Table IV that there were many such cases—there were no striking changes in the morphology of the cells. Virtually no abnormal or immature cells were found in any case. Changes in size and shape of the cells when they did occur were very minor and regarded as having no important significance.

*Carbon Monoxide Content of the Blood*—Carbon monoxide determinations were made on whole blood by the Van Slyke method. The findings are presented in Table V. Since the workers examined were not exposed to very high concentrations of carbon monoxide gas the carbon monoxide content of the blood was gener-

ally speaking not high. It might be well however, to bear in mind the fact, that there is a considerable variation in the carbon monoxide content of the blood in these workers from time to time during the day, corresponding to the variations in the concentration of carbon monoxide in the air in different parts of the workroom. With the present lack of proper means for directly removing the exhaust from cars which are being tested, workers in most garages are subjected at different times of the day to really high concentrations of the gas. This is particularly the case when they are working in the immediate vicinity of a car which is exhausting a considerable quantity of carbon monoxide while being tested. Several of the workers who complained of palpitation of the heart and faintness claimed that they felt it only at such times.

In the present series, all of the cases were those of chronic exposure to the gas. The duration of total exposure varied from 1.5

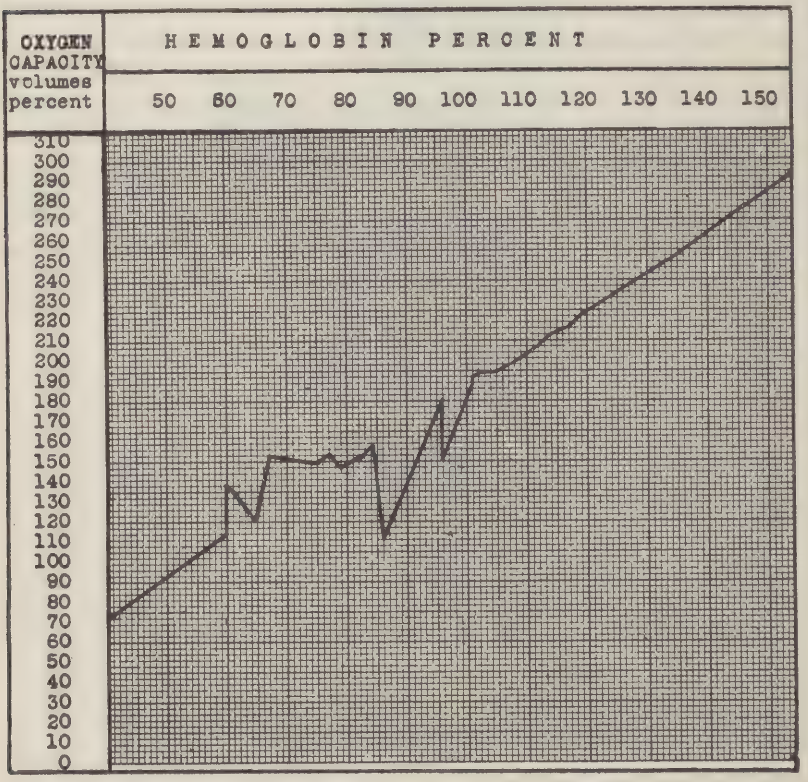



**Chart 6. Curves for Calculating the Per Cent of Carbon Monoxide from the Per Cent Blood Saturation**

to 25 years, and the duration of the exposure on the day of examination varied from 30 minutes to two hours—subject to variations in degree.

*Oxygen Capacity and Oxygen Content of the Blood*—Determinations of the oxygen content of the blood were made on venous blood. The oxygen capacity of the blood was calculated from its hemoglobin content, since it was considered that arterial blood is normally practically 100% oxygenated. In normal arterial blood therefore the oxygen capacity approximately equals the oxygen content. The difference between the oxygen contents of venous and arterial blood generally represents the oxygen usage by the tissues. The oxygen capacity therefore of venous and arterial blood tends to be the same, though their oxygen content will differ, depending upon the amount of oxygen which has actually been used up by the tissues in the course of their metabolism.

The oxygen content of the blood of the men examined ranged from 2.4 volumes per cent to 10.8. The normal was approximately 19 per cent. The average content of these men was found to be 5.8 volumes per cent, or a little more than one-fourth the normal oxygen content of the blood, showing a very striking anoxemia. There appeared, however, to be no correlation whatever between the precise quantity of carbon monoxide in the blood and the oxygen content found.

*Oxygen Unsaturation*—The oxygen unsaturation of the blood was calculated as the difference between the oxygen capacity (calculated from the hemoglobin content of the venous blood) and the oxygen content of the venous blood.

The most striking blood finding, as may be seen in Table V, is the great oxygen unsaturation of the blood. Taking 2 to 4 to be the normal oxygen unsaturation for adults, all except two cases out of 27 were found to be abnormally high, ranging from 4.7 to 25.1; 20% of them being over 8. This increased difference between arterial and venous oxygen content was not due to an increased oxygen content of the arterial blood (that is to say, an increased oxygen capacity) but rather to a marked diminution in the oxygen content of the venous blood. Thus, instead of the normal 18–20 volumes per 100 of oxygen in the venous blood, these cases range from 7.9 to 10.1. In other words, a true anoxemia was observed in these chronic cases which was out of all proportion to the degree of saturation of the blood with carbon monoxide. The degree of oxygen unsaturation was apparently due primarily to the fact that the oxygen content of the venous blood was unusually low, while

the hemoglobin, and therefore the oxygen capacity, was approximately normal.

It will be remembered that in exposure to carbon monoxide a certain amount of oxygen in the arterial blood is displaced by carbon monoxide, and so the oxygen content of the arterial blood is lower than normal. The oxygen content of arterial blood in these cases is no longer equal to its oxygen capacity but is equal to the oxygen capacity minus the carbon monoxide content of the blood.

Calculating the oxygen unsaturation after subtracting the carbon monoxide content of the blood from the oxygen capacity of the arterial blood, it is still found to be large. See Table V.

In other words, the oxygen content found in these cases appears to be lower than one would be led to expect from the amount of carbon monoxide which was absorbed into the blood.

This might be regarded as evidencing the fact that the tissues were probably using an abnormally large amount of oxygen, under these conditions. Actually, this might be anticipated because during the period of recovery after asphyxia there is an abnormally large consumption of oxygen by the muscles. In a situation of low-grade concentration of carbon monoxide in the air, where oxygen and carbon monoxide are constantly circulating through the blood, the individual muscle cells might be regarded as alternately receiving and being deprived, in turn, of their normal oxygen quota. As a result the muscles as a whole might be thought of as being in a more or less continual state of recovery from partial and temporary asphyxia. During recovery there appears to be an increased demand for oxygen on the part of the muscle in order to oxidize the excess lactic acid which has accumulated during the period of asphyxia.

These findings throw further light upon the relation of carbon monoxide absorption to exercise. If one considered the increased demand for oxygen necessitated by the period of recovery from asphyxia, and then adds the increased oxygen demand resulting from muscular exertion, one can more fully appreciate that both these demands for oxygen *in an individual with a marked oxygen unsaturation* of the blood might very readily result in an acute attack of carbon monoxide poisoning, even after the victim has been removed from the actual exposure.

*The Carbon Dioxide Content of the Blood*—The carbon dioxide content of the blood was determined by the Van Slyke method on whole bloods taken under albolene. It may be seen by reference to Table V that in 8 out of 27 cases the $CO_2$ content

of the blood was less than 50 vols. per 100. (Values from 25.2 to 49.5). This again is a somewhat surprising finding in cases where the concentration of carbon monoxide in the blood is relatively so low. However, in view of the fact that there was an increased respiratory rate, this may be looked upon as a manifestation not of a true acidosis but of a compensated alkalosis; an acapnial process, perhaps, such as Haggard and Henderson have described as occurring to a more marked degree at high concentrations of carbon monoxide absorption, (over 50%). In their cases hyperpnoea occurs which results in a blowing off of $CO_2$. This tends towards an alkalosis. In an attempt to compensate for this the kidneys excrete alkaline urine ($Na_2HPO_4$, in excess of $NaH_2PO_4$) which results in a loss of base from the body and gives the same end result (low $CO_2$ and low total base) as in a condition of acidosis.

*Serum Potassium*—The nervous irritability which was regularly found to be associated with the carbon monoxide headache, suggested still another line of investigation. Jacques Loeb and others demonstrated that increased nerve irritability frequently accompanies an increase in the potassium content of the blood serum, and it has been noted that in new born infants who give increased electrical reactions, the potassium content of the serum is almost invariably high.

In view of the apparently intimate relationship which has been found to exist between the relative concentrations of the inorganic ions, potassium, sodium, and calcium, in the serum, and nervous manifestations; and in view of the possibility of some change in the concentration of these ions accompanying the shift in acid base equilibrium which is thought to follow exposure to carbon monoxide gas, it was felt that some interest might attach to a study of the concentrations of these ions in the present instance. That these ions cannot permeate the normal cell wall has been shown by Peters, Eiseman and Hsieu Wu.

Potassium was the first of the inorganic ions selected for the study. The analyses were made upon whole blood, taken from the vein—every precaution being taken to prevent hemolysis of the serum. The Kramer and Tisdall method was used for the analyses which were made within a very few hours after the bloods were collected. In spite of these special precautions to prevent hemolysis, 11 out of 22 bloods taken were definitely hemolysed. In 9 cases, however, where the question of hemolysis was definitely eliminated, the potassium content of the serum was normal.

## Summary

Reviewing the results of physical examinations and blood tests, attention is especially directed to the following:

1. Physical examinations and blood counts were made on 50 garage workers who were chronically exposed to relatively small concentrations of carbon monoxide gas. There were, therefore, no cases of acute asphyxiation in the series.

The blood count included:

a. Hemoglobin determination.
b. Red blood cell count.
c. White blood cell count.
d. Differential count.
e. Examination of the morphology of the cells.

2. In the case of 27 men, the following additional blood determinations were made:

a. Carbon monoxide content of blood
b. Oxygen content.
c. Oxygen capacity.
d. Oxygen unsaturation.
e Carbon dioxide.
f. Serum Potassium.

4. The duration of exposure of the men examined varied from 1½ to 25 years.

5. On the day of examination the exposure varied from approximately 30 minutes to 7 hours.*

6. The carbon monoxide content of the air ranged from 2.3 parts per 10,000 in those parts of the room not immediately adjacent to cars giving off an exhaust, to 11 parts per 10,000 immediately behind cars which were exhausting gasoline fumes. The men were going about from one part of the room to another so that they were exposed to varying concentrations of the gas at different times in the day. They were in the habit of going out of doors occasionally to get some fresh air.

The symptoms of which the men complained, and the incidence of such symptoms, were as follows:

a. Headache, 74%
b. Dizziness, 50%.
c. Burning of the eyes, 32%.
d. Gastro-intestinal disturbances, 24%.
e. Dizziness, 28%.
f. Faintness, 12%.
g. Insomnia, 20%.
h. Cardiac symptoms, 18%.
i. Mental depression, 12%.
j. Irritability, 30%.
k. Ringing in the ears, 10%.
l. Cough, 28%.
m. No symptoms, 8%.

The majority of the symptoms are referable to the nervous system, and the carbon monoxide headache predominates. All of the symptoms listed are those usually regarded as prodromal or warning symptoms which ordinarily precede acute asphyxia.

* Causes for possible inaccuracy in this have been discussed. See p. 22.

8. The carbon monoxide headache, as experienced by the men examined, appeared to be extremely intense and incapacitating, characterized by pounding and throbbing in the temples, and at the base of the skull; increased by bending, and frequently associated with nausea and dizziness. Its onset may occur hours after removal from exposure to the gas and indeed after all carbon monoxide has undoubtedly been eliminated from the blood. It may last a great many hours and in some cases may necessitate absence from work for a day or more. It is recognized as the chief cause for loss of time among garage workers. The explanation of the late onset of the headache would appear to lie in the fact that it is caused by an increase in intra-cranial pressure which tends to become progressive during the period of recovery from the exposure.

9. Marked pallor, noted in 76 per cent of the cases, did not appear to have any definite relation to the hemoglobin content of the blood. In 26 cases pallor was observed where the homoglobin was 80% or higher. This may be due to a vaso-constriction of the superficial capillary circulation, somewhat analogous to that found in persons exposed to lead.

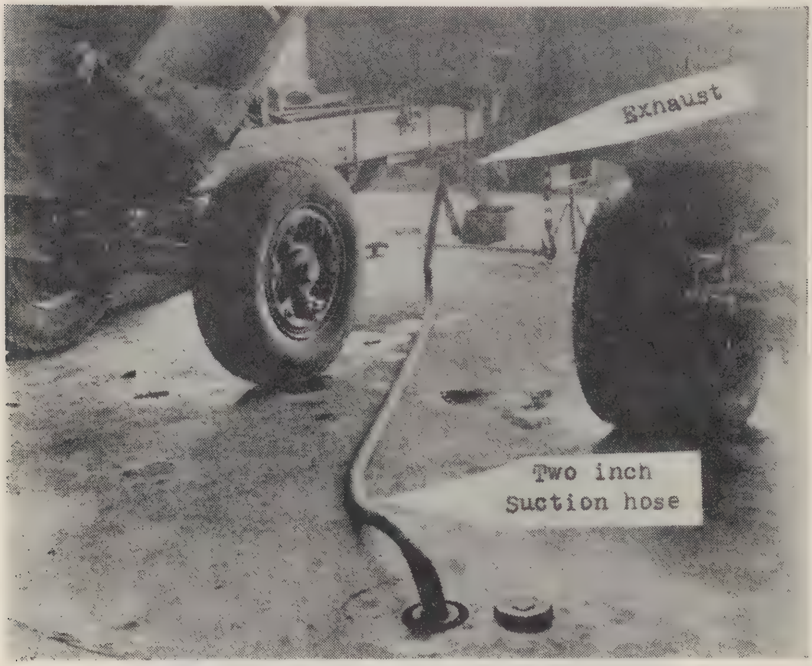
**Individual Pipe Connection to Muffler Exhaust in Garage**

10. The cardio-vascular system was essentially negative on clinical examination, except for almost uniformly high pulse rates. Nine of the workers examined complained of cardiac disturbances as follows:

a. Palpitation on exertion, 5 cases.
b. Pre-cordial pain, 2 cases.
c. Shortness of breath, 2 cases.

The negative clinical findings suggest that it might be of considerable interest to investigate the hearts of men chronically exposed to small concentrations of carbon monoxide gas by means of the electro-cardiograph. This is of especial interest because of the importance of cardiac sequelae following carbon monoxide poisoning.

The pulse rate was almost uniformly high. It ranged from 75 beats per minute to 130; the average being 101.84, as compared with the normal of 72. In 68% of the cases the pulse rate was above 90. The significance of a rapid pulse rate which is maintained over a long period of time from the standpoint of cardiac strain is commented upon.




**State Highway Garage, New Hamburg, N. Y.**

The blood pressure findings were generally speaking of normal range. In some cases there appeared to be a marked fall in the systolic pressure in the course of the day, while in others there was a tendency to a distinct rise. This is probably due to individual differences in cardiac response not merely to the continued maintenance of high pulse and respiratory rates, but to abnormal biochemical relations in the body incident to exposure to exhaust gas. The individual daily variations were higher than had been observed in previous investigations.

11. The fact that 14, or 28%, of the workers complained of cough was of especial interest, in view of the fact that pneumonia is an important sequelae of carbon monoxide poisoning. Examination of the lungs was negative, however. Cough was so frequently associated with burning of the eyes that it was thought that it might be a manifestation of an allergic reaction (hyper-sensitiveness to exhaust gas) of an asthmatic character, rather than due to any direct effect of carbon monoxide per se upon the respiratory tract. No conclusion, however, could be reached in the matter on the basis of the present investigation.

The respiratory rate was high in most cases, 42 of the men examined having a respiratory rate above the normal 22. The average for the series was 25.88.

12. Examination of the nervous system was confined to the following:

a. Tremor, either of the hands or of the tongue.
b. Activity of the reflexes as measured by the knee jerks.
c. General emotional and nervous instability of the workers.

Tremor was found in 46% of the cases and hyper-active knee jerks in 40%. The workers, generally speaking, were observed to be more suggestible and less stable emotionally than workers examined in other occupations. Marked increase in nervous irritability was very common following even short exposure to higher concentrations of the gas (as when repairs were being made on a car which was discharging in the immediate vicinity of the worker).

13. The red blood cell counts and the hemoglobin determinations showed distinct differences in individual reaction. Some appeared to compensate fairly adequately for the oxygen deficiency by an increase in the red blood cells, and hemoglobin, thus acquiring an actual increase in the oxygen receptors of the blood; while others did not seem to succeed in making this compensatory adjustment, but maintained an approximately normal blood picture. In still

other instances, there was a tendency to a reduced red cell count with a reduced hemoglobin content, all of which would necessarily so decrease the oxygen carrying power of the blood as to make any further displacement of oxygen by carbon monoxide distinctly hazardous. Symptoms were not confined to those workers who were unable to compensate by an increase in the blood cell count. The effect of contaminants in modifying the reaction of individuals to carbon monoxide exposure must not be lost sight of.

14. Further investigation of the blood showed a degree of oxygen unsaturation apparently out of all proportion to its carbon monoxide content. This would seem to indicate that in all probability under conditions of carbon monoxide exposure the tissues tend to consume a greater quantity of oxygen than under normal conditions. The analogy between the ordinary increase in oxygen required by muscles recovering from acute asphyxia, and the situation of these men whose tissues are in a process of continual recovery from partial asphyxia, so to speak, was pointed out as a possible explanation of the increased oxygen consumption.

15. This increased oxygen consumption in an individual whose blood is characterized by a marked oxygen unsaturation, to begin with, is important in throwing light upon the ease with which he may succumb to asphyxia on exertion—a condition creating a still further demand upon his oxygen reserve.

16. Garage workers are exposed to a mixture of gases in exhaust gas, most important of which is carbon monoxide. There is reason to believe that the normal reaction to pure carbon monoxide is modified in important ways by the presence of contaminants.

# CARBON MONOXIDE IN THE HAT INDUSTRY—I

MAY R. MAYERS, M.D.

Exposure to carbon monoxide gas in workrooms where there are large numbers of small gas-heated appliances in operation is very great at this time of year. In industries where large gas-heated appliances are used, they are usually installed with considerable care, and overhauled from time to time for purposes of efficiency. The greatest hazard to health from constant exposure to carbon monoxide gas in the lower concentrations, therefore, occurs in industries such as the hat industry, where the appliances used are relatively small and inexpensive, and the attention paid either to their installation or upkeep often is insufficient. In fact, this industry illustrates so well the problems common to such a large number of industries as to warrant a rather detailed account of the situation as found there on recent investigation.

Hat manufacturing in New York City now occupies a relatively small number of high loft buildings in and about the West Thirties. There are, indeed, some sixty odd hat factories at present within a radius of only a very few square blocks in this district. With this concentration of the industry into so limited an area, came a sudden demand for quantities of gas far in excess of that contemplated when some of the loft buildings were erected. With this new development came also the introduction of large numbers of gas-heated appliances—gas-heated hat presses of various sorts, and gas-heated boilers for making steam—sometimes in the hands of persons ignorant of their proper operation and of the dangers latent in improper operation.

In the early days, the women's hat industry was in the hands of a few stable well established concerns. Within a few years, however, the situation was practically revolutionized, and at the present time, the industry is in the hands of a large number of very small hat manufacturers, all in the keenest competition with one another. Many more hats are being produced than ever before. There has been a corresponding increase in the total quantity of gas equipment in use. The outlay is small; a few sewing machines and hat presses are readily obtainable on credit. Almost any employee can go into the business for himself, if he is enterprising, and if he has no luck he can give up his shop, and go to work for some one else again the next season. The more or less primitive industrial organization now obtaining in the hat industry is, therefore entirely analogous to what is found in many other industries where a small initial investment is required, and where competition is intense. The work is highly seasonal, and subject moreover to all sorts of fluctuations of style, so that in the hectic rush of production often little thought appears to have been given to considerations of efficiency of operation of the machines or to health maintenance of the workers.

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## Gas Leaks

The uniformity of equipment and of procedure in large and small workshops alike, is very interesting. In general they tend to buy their hat presses and their steam boilers from the same manufacturers—apparently with little regard to safety approval by the American Gas Association Laboratories. They even buy their rubber tubing for use on the presses from the same wholesale dealers, seemingly through ignorance of, rather than in defiance of, the regulations specifically prohibiting the use of any flexible tubing except that approved by the City Department of Health.

As one walks alongside of the presses—whether they are actually in operation or not—there is frequently a distinct odor of gas. If one investigates further, one is surprised and disturbed at the number of places from which small amounts of gas are escaping, due to faulty connections, worn-out tubing, etc. When the employers, or any of the workers in the vicinity are questioned they usually express very genuine surprise. They have all become so accustomed to the smell of gas they do not appear to notice the leaks. Those most anxious to cooperate with the investigator usually strike a match at once, and begin running it along some of the gas connections, and especially the rubber tubing which has been the object of special criticism. Sometimes the leak is so large they get a light. Then they hasten to put some kind of a patch over it, and look entirely self-satisfied. More often there is no light obtained because the leak is too small, and they return from their efforts looking distinctly injured, for they believe that they have thereby proved conclusively that no leak exists. One is quite as much impressed with their desire to be informed, and their willingness to cooperate as one is with their ignorance. They all know that gas is dangerous and they are entirely prepared to do what is necessary to keep their workrooms free from gas—provided that their competitors are required to comply with the same regulations. They know far less about the dangers of carbon monoxide poisoning which results from incomplete combustion of the gas in improperly designed gas-heated appliances; and those in need of cleaning and repair. They require a great deal of education. The industry must be treated as a whole, however. No single plant can afford to adopt standards which are higher than those of its competitors.

## Poor Combustion in Hat Presses

The hat presses or hat blocks have passed through many stages of development over a period of many years. Though originally designed for steam, they were later converted to burn gas. Poor combustion in the operation of these presses is due, in large measure, to the fact that in the process of conversion the steam chamber was never properly modified for its new use as a combustion chamber for gas. Various types of gas installations have been

tried, but none of them has proved entirely satisfactory from the standpoint of good combustion.

The Consolidated Gas Company and the American Gas Association have long been interested in this problem, and they have been instrumental recently in assisting in the development of a modification of the heating elements in the hat block, now in use, so that its carbon monoxide production may be maintained within safe limits. Only 1 part per 10,000 of carbon monoxide was found in tests of flue gases given off by these machines—which means that there would be far less than that amount in the air of the workroom as a whole.

In the case of all new installations, it is suggested that hat block manufacturers make alterations in the original castings so that the ordinary ring burners may be used for heating. Where old installations are used, however, they should be modified along these newly developed lines, so as to make their operation safe. A helpful discussion of the problem and its solution as presented by the American Gas Association follows:

"There are several makes of hydraulic hat blocks used in the New York District, but they are all practically identical in construction. At the top of the casting which is to be heated, there are three radial space 1½ inch openings (formerly steam inlet connections) which are to be used as flue outlets, and at the bottom there are six 1½ inch holes (formerly condensation outlets) which can be used for firing the blocks. (See Fig. I)

"The burner equipment originally used on the blocks and which is still in use in a great many cases consists of two crude pipe burners with fixed orifices and primary air openings, and a single large port. With these burners combustion is poor, and the flames float. In 1931 this situation was remedied by heating the blocks with six barber jets connected to a circular manifold at the base of the castings. However, this burner has not been widely adopted, and now to encourage the safe utilization of the gas, a new burner has been developed which is an improvement over this former burner from the viewpoint of heat distribution.

"The new burners consist of a round burner-casting attached by screws to the bottom of the block, which acts as a manifold for six blue flame jet type burners, one jet projecting into each of the six condensation holes. The tips of these jets are constructed as shown in Fig. I, so that the flame produced is flat and fan shaped, and can fit into the steam jacket space without impingement. The jets have No. 58 orifices and the normal consumption of the burners is about 25 cu. ft. per hour.

"Because of the fact that the condensation outlets on all the blocks are not exactly in the same position the burner castings are blank and have to be tapped (standard ⅛″ pipe thread) on the district in accordance with the dictates of each particular job. This can be done with a hand drill and the proper size tap. Holes also have to be drilled and tapped in the bottom of the block casting for the screws that hold up the burner assembly.

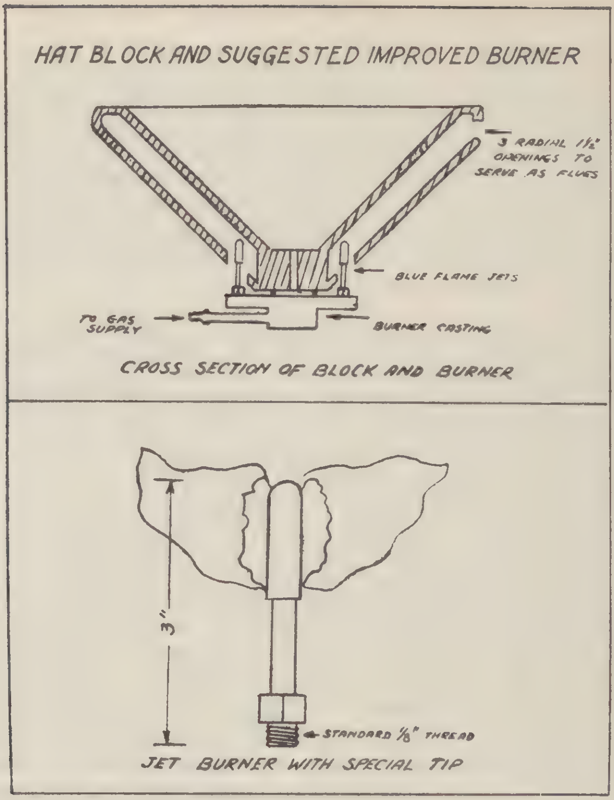


**Fig. I**

Since the entire block casting moves up and down in action the gas connection is made by means of a flexible tubing.

"It is absolutely essential that the three flue outlets be left open and unrestricted, and that the burner tips be placed in their proper position. That is, there should be no impingement of the flames on the casting."

## Steam Boilers

The steam boilers used in the hat industry are heated by gas, and are the other important source of carbon monoxide production in these workrooms. All such boilers are required to be flue-connected at the present time so that all flue gases may be properly exhausted at their source. Frequently, fans are not kept in operation, and windows which are counted on for ventilating boiler rooms are kept closed. The exhaust system provided for all such steam boilers should be kept in good repair and must be in operation at all times when the steam boilers are in use.

## Summary

Both employer and employee must be educated to an understanding of the elementary principles of safety in the use of gas equipment. Practically it has always been found necessary to point out the following:

a. Injury to health may result either from leaking gas, or the production of carbon monoxide gas by poorly designed ill-kept equipment.

b. Small leaks incapable of lighting when a match is applied are just as important as the larger ones.

c. The fact that no one smells a leak is of no significance because it is easy to become accustomed to the odor.

d. Improvised patches on gas leaks are no remedy. They must be properly repaired.

e. Appliances must be kept cleaned and free from soot in order to prevent unnecessary production of carbon monoxide poisoning in their operation.

f. The free maintenance service of the Gas Company should be called upon frequently, and their recommendations as to replacement of worn parts should be promptly complied with.

g. A yellow flame or one which appears to spread or float is dangerous.

h. If the weather is cold, and the presses are cold, a dangerous amount of carbon monoxide gas may be released when the flame under them is started. A gas flame from an improperly adjusted burner impinging on a very cold surface may release a considerable amount of carbon monoxide gas. It is well to start the gas some time before the workshop opens so that the room may be thoroughly ventilated before workers are permitted to enter.

i. All flexible tubing must be of the type approved by the City Department of Health.

j. Steam boilers should always be properly vented. Exhaust fans must be in operation at all times when the boilers are in use.

Several times in the last few years workers in establishments of the general type above described have suddenly been overcome by carbon monoxide gas. All of these occurrences were in winter and on Monday mornings which gave them something of an air of mystery until investigation revealed the following: The building is cold after Sunday with the heating plant a little late getting started. The workers come in, and because of the cold, do not open any windows. Frequently, in order to warm the room quickly, every gas-heated appliance that is available is rapidly lighted—even those which will later be turned out as not needed for the day's work. Steam boilers are started with windows closed and

fans not operating, in order to get heat for the workroom. The result is an abnormally great production of carbon monoxide gas in the room—partly because of the flames impinging on unusually cold surfaces: partly because more than the usual number of appliances are put into operation; partly because many of the appliances are of poor design and in poor repair, and partly because exhaust systems in connection with the steam boilers have not been started promptly. All of this added to the "normal" leakage of raw gas in such establishments and the fact that all windows are kept tightly closed explains this Monday morning phenomenon.

# CARBON MONOXIDE IN THE HAT INDUSTRY—II

MAY R. MAYERS, M.D. AND WM. J. BURKE, CH.E.

The hat blocking industry in New York City has for some time presented a special problem of industrial hygiene. Every winter, despite the best efforts of the Department of Labor, a certain number of workers have been overcome by carbon monoxide gas. The peculiar working conditions in this industry which are in part responsible for this situation were reviewed in the previous article which directed attention to the lack of properly designed gas-heated equipment in use in the industry; the need for better upkeep and cleanliness of equipment, and the prevalence of gas leaks through leaky rubber tubing and leaky gas connections.

It is the purpose of the present article to discuss in somewhat greater detail the special engineering problems presented by the gas-heated hydraulic hat blocking machines which are perhaps the greatest single source of carbon monoxide production in the workrooms in this industry.

Women's felt hats are pressed, by means of this machine, in an aluminum mold. (Fig. I.) The mold, in turn, is placed into a bowl-like conical-shaped casting which forms the base of the machine. The molds receive their heat by conduction from the castings which in turn may be heated either by steam or by gas. Although originally designed for the use of steam, in the Metropolitan area the presses have been converted so that they may be heated by gas.

In the process of conversion, the original steam jacket is used as a combustion space for the gas. The entire machine, however, is not re-built, because it is the practice in the trade to sell used presses over and over again, and it must always be possible to operate them with steam if necessary. All attempts at re-design for adapation to the use of gas have therefore been directed toward the installation of a proper type of gas burner without attempting in any way to modify the original casting. There are, at present, three distinct types of gas burners in use known respectively as (A) the Pipe Stem Burner (B) the Barber Jet Burner; and (C) the Bat Wing Burner. These represent progressive attempts on the part of manufacturers to reduce the amount of carbon monoxide produced in the operation of the presses. Before discussing them in detail, however, it might be well to review briefly some of the principles underlying proper gas combustion—particularly as applied to this type of gas-heated appliance—because the large quantities of carbon monoxide which it produces are chiefly due to defects in design resulting in incomplete combustion.

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## Composition of Illuminating Gas

The combustion of manufactured gas varies somewhat with the process of manufacture. The composition of gas supplied to the City of New York is presented below in Table I. Listed after the various components (with the exception of carbon dioxide and nitrogen, which are not combustible), is the amount of oxygen necessary for the complete combustion of one cubic foot of each gas. It will be observed that complete combustion of one cubic foot of illuminating gas as a whole requires 0.945 cubic feet of oxygen or approximately, 1 cubic foot of oxygen. For practical purposes, we may assume that air is composed essentially of 20.91% oxygen and 79.09% nitrogen by volume—the other gases being present in too small amounts to play any role in the chemical reaction. The amount of air required therefore to provide the necessary oxygen is approximately 4.79 cubic feet per cubic foot of illuminating gas. This gas has a heating value of 550 British Thermal Units per cubic foot. (British thermal unit is the amount of heat required to raise the temperature of one pound of water one degree Fahrenheit—from 39°–40°.)

Table I. Present Composition of Gas

| | Percent | Oxygen required per cubic foot of gas |
|---|---|---|
| Carbon dioxide | 4 | ............... |
| Ethane | 6.0 | 0.1800 cubic foot |
| Benzene | 2.6 | 0.1950 cubic foot |
| Oxygen | 1.0 | 0.0100 cubic foot |
| Carbon monoxide | 24.9 | 0.1245 cubic foot |
| Hydrogen | 28.7 | 0.1435 cubic foot |
| Methane | 15.6 | 0.3120 cubic foot |
| Nitrogen | 17.2 | ............... |
| | | 0.9450 cubic foot |

Since, as will be observed from the above table, carbon monoxide is itself one of the important constituents of illuminating gas, it is clear that the possible sources of carbon monoxide in the air of any workroom where gas-heated appliances are being operated are two-fold: (1) Leakage of unburned illuminating gas from defective piping and loose connections—a subject discussed in some detail in the previous article—and (2) the liberation of carbon monoxide in the operation of these appliances resulting from incomplete combustion of the supplied gas, and due to poor mechanical design. The present article is directed primarily to a consideration of the latter.

Fundamental to the design of any atmospheric gas burner is provision for adequate oxygen supply so that a proper quantitative relationship may be maintained at all times between the air and the gas to be burned.

**Fig. I**
**Hydraulic Hat Press Open, Showing: Aluminum Mold, Front Port Hole Open; Burners Entering Condensation Holes.**

The oxygen used comes from two sources, known as primary and secondary air.

The primary air, which amounts to from 50-60 per cent of the total air requirement is drawn into the gas-mixing chamber from a small orifice provided at the base of the burner. This mixes with the gas as it issues from the burner.

The secondary air is the air which immediately surrounds the flame.

While theoretically about one cubic foot of oxygen is required for each cubic foot of illuminating gas, in practice it has always been found necessary to provide a considerable excess of oxygen in order to be assured of good combustion. In the case of the hydraulic hat press, it has been estimated that this excess should be as high as 25 per cent due to the fact that the only contact of the secondary air with the gas flame is at its periphery.

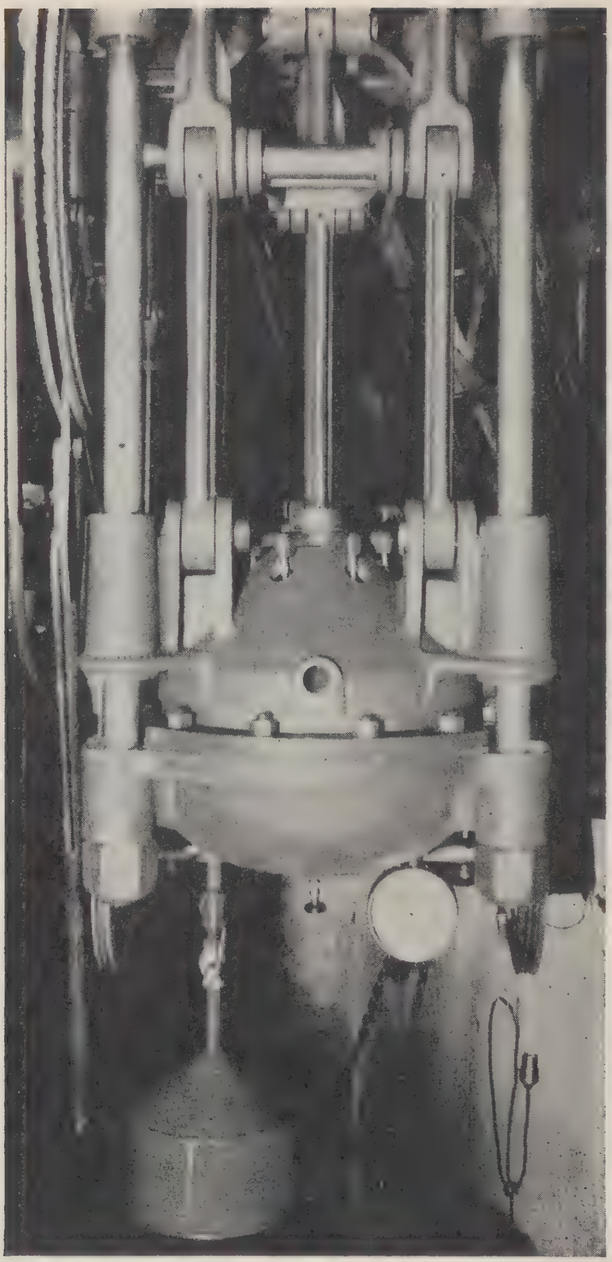

Fig. II
Hydraulic Hat Press Closed, Showing: Front Port Hole Open; Burners Entering Condensation Holes.

## Flue Gases

The combustion of one cubic foot of gas results in the production of six cubic feet of flue gases. When illuminating gas is completely burned, the resulting flue-gases will not support combustion, being composed chiefly of carbon dioxide, water vapor and nitrogen. When combustion is incomplete, however, flue gases always contain carbon monoxide—the amount varying inversely with the completeness of combustion. The combustion chamber should always be large enough to insure against an unduly great accumulation of flue gases in the immediate vicinity of the flame, for such an accumulation tends to dilute the local air supply to the gas burner, thus reducing the amount of oxygen available for normal combustion.

Moreover, if the combustion area is too small, efficiency is materially reduced, because for each cubic foot of carbon monoxide which fails to be burned to carbon dioxide, there is an estimated loss of 347 British Thermal Units.

The combustion chamber must also be kept clean. An accumulation of carbon in this chamber not only reduces the size of the combustion area, but when it becomes red hot tends to reduce carbon dioxide to carbon monoxide when a flame impinges against it.

The flame furthermore should never be permitted to impinge upon a cold surface. The ignition temperature of gas is approximately 800°F. Impingement on cold objects tend to reduce this temperature below the ignition point and the liberation of carbon monoxide results.

The several makes of hydraulic hat blocks now in use in the New York district are practically identical in basic construction. In all cases the casting, around which the rest of the machine is built, so to speak, has three circular openings in its periphery, each about one and one-half inches in diameter. These were formerly steam inlet connections and are now being used as flue gas outlets. In addition, there are six holes of one and one-half inch diameter (formerly condensation outlets) at the bottom of the casting which may be used for firing the blocks. (Fig. I and II).

## "Pipe Stem" Burner

In the initial attempt to convert these presses for the use of gas instead of steam, two crude pipe burners with fixed orifices and primary air openings were affixed to the casting. This came to be known in the trade as the "Pipe Stem" burner. With this type of burner, combustion is very poor indeed; there is a tendency for the flame to "float" and large amounts of carbon monoxide gas are produced which pour into the room through the three circular openings in the casting above described. (See "Test" Table). Incidentally, the flue gases coming out of these openings contain so much carbon monoxide that they themselves are capable of supporting combustion, and one may observe two tongues of flame shooting out of the two side portholes when the machine is in operation.

The third flue outlet (Fig. I) which is located directly in front of the worker who operates the machine is always kept tightly closed by means of a metal fitting. It would otherwise be literally impossible for the operator to stand in front of the machine. Though seemingly a simple remedy, this practice of keeping the front porthole permanently closed, is in reality very dangerous, because it acts to further reduce the amount of air available for combustion, and very greatly increases the total amount of carbon monoxide given off into the room through the two remaining outlets. It has the immediate advantage, however, that the flue gases do not discharge directly under the operator's nose; and that he is protected from having his clothes singed by the tongue of flame shooting out through the porthole in front of him. Incidentally, in addition to all else, this type of burner gives off a most disagreeable odor which is quite independent of its carbon monoxide production, since the latter gas is odorless. The air analyses given below which were made with this machine in actual operation show clearly what large amounts of carbon monoxide gas are released into the workroom. (See Table II)

TABLE II. TESTS OF TYPES OF BURNERS

| | Air sampling location | |
|---|---|---|
| Type of Burner | Air portholes | At breathing level of operator |
| II. Pipe Steam Burner................. | Over 15.0 | Over 15.0 |
| II. Barber Jet Burner................. | 5.5 | 7.0 |
| II. Bat Wing Burner.................. | 0.3 | 0.0 |

At the present time, this particular type of "Pipe Stem" burner is in use in the great majority of hat factories in the metropolitan area, where hydraulic hat presses are used. Since in most of these plants, a great variety of operations are carried on in a single room, exposure to carbon monoxide gas in injurious amounts is not confined to the operator of the hat press alone but indirectly affects all of the other workers in the room.

### "BARBER JET" BURNER

In 1931 an attempt was made to improve this situation, by the introduction of an entirely different type of burner known as the "Barber Jet" burner. A circular manifold is attached to the base of the casting to which six "Barber Jets" are connected. Air analyses made with this type of hat press in operation show that while it is an improvement over the original type of burner, it does not offer adequate protection against the production of carbon monoxide gas in amounts dangerous to health. (See Table II) Here again, it is the practice to close up the flue opening in front of the machine, because experience has shown that the flue gases rising into the face of the worker tend to give him a

headache. These flue gases, however, do not appear to contain enough carbon monoxide gas to support combustion as in the earlier machine and consequently one does not observe any actual tongue of flame shooting out of the portholes when this machine is in operation. The odor given off by this machine is nevertheless about as bad as in the case of the earlier type.

On the whole, the "Barber Jet" burner has not been widely adopted in the industry. Its advantages, though real enough, were apparently not sufficient to impress purchasers. Also, because of the highly competitive nature of the industry, and the small initial investment required to go into business, there is a tendency to buy used hat presses on time payments. This seriously interferes with the introduction of a new type of machine.

### "Bat Wing" Burner

The economy of operating a machine designed for good combustion has been graphically illustrated in the case of the third type of burner which has been recently developed, known as the "Bat Wing" burner. (Fig. III) The hat press to which this burner is affixed is identical with that formerly used. The burner itself is very like the "Barber Jet" burner above described except that the tips of the jets are so constructed as to produce a flat and fan-shaped flame, and they fit into the steam jacket without impingement. The jets have No. 58 orifices; and, as has been pointed out, the consumption of gas is cut down by one-third as compared with either of the older machines. Instead of requiring three gas jet connections to the main gas supply, to operate the machine only two are needed—thus cutting down the gas consumption to approximately two-thirds of that used by either of the first two machines. Its normal gas consumption is about 25 cubic feet per hour. The additional $20 investment required to equip the older machines with the new burner would obviously be more than refunded in a relatively short time through a reduction in the gas bill.

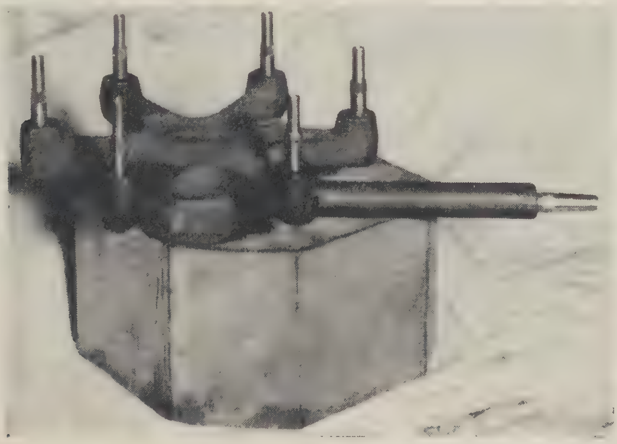

**Fig. III. "Bat Wing" Burner**

The new burner as developed appears to be perfectly safe judging by all air analyses made both in the field and in the laboratory. Results of air tests made in the field are presented below. Moreover, this type of machine can be comfortably operated with all three portholes open—as they should be in all of the gas-heated machines. In fact all portholes were found to be open when this type of machine was inspected in actual use. The carbon monoxide content of the flue gases was found to be well within safe limits; there was no unpleasant odor in the room; and no flame could be observed coming out through the portholes, as when the "Pipe Stem" burner is used. In operating this type of machine there is apparently no desire on the part of the worker to close up the porthole directly in front of him.

Air tests were made on all three types of machines in the field to determine the precise amount of carbon monoxide gas which they produce. They were tested under actual operating conditions, so that the first two types were tested with the front portholes closed—as is the custom in the industry—and the third was tested with all portholes open since this type of press is actually operated in this manner. In each case the first sample of air was taken for analysis immediately outside the flue outlet of the machine, while it was in operation, in order to determine the carbon monoxide content of the flue gases. The second air sample was taken at the breathing level of the worker who was operating the machine. The results are expressed in parts of carbon monoxide per 10,000 parts of air in Table II.

## Conclusions

Gas-heated hydraulic hat presses are now available to the hat industry which are safe from the standpoint of carbon monoxide production, and economical from the standpoint of gas consumption. In order to insure their safe operation in the plant the burners should not be altered after installation, as the ratio of gas and air for proper functioning of the burner in the space provided has been accurately determined to obtain the most efficient and safe burning of the gas. Machines should moreover be kept clean (free from carbon) and in good repair at all times. The gas company should be called upon from time to time to service all presses; and recommendations as to replacement of worn parts should be promptly complied with. The gas company service is free of charge.

The proper design of atmospheric burners for the efficient burning of gas in an enclosed combustion area such as is found in the hydraulic hat presses is a complicated engineering problem. Burners, when properly designed will produce a maximum of heat with a minimum of gas consumption and will give off very little if any, carbon monoxide gas. Properly designed gas-heated appliances are therefore not merely safe but are more efficient and, therefore, more economical to operate. Once again economy in operation yields safety and health protection to the worker.

# CARBON MONOXIDE IN FOUNDRIES

William J. Burke, Ch.E.

Persons conversant with foundry operations are familiar with what is known as the "gassing stage" which follows the pouring of molten metal into sand molds. During this stage, tongues of flame may be seen shooting out from the sides of the molds, and smoke and irritating fumes are given off in considerable quantities. Air analyses have shown that where such smoke is due to the sea coal, or pitch used in the mold, sulphur dioxide may also be present.

Materials used as binders in core making may also be responsible for the production of irritating gases in the pouring operations. For example, when linseed oil is used as a binder, acrolein fumes may be produced which are extremely irritating to the eyes of the workers exposed. Linseed oil and corn starch (trade name Kordek) are the substances most commonly used as binders. However, linseed oil may be dissolved in mineral oil for the purpose, or a mixture of cornstarch and rosin may be used in addition to the linseed oil. Irritating fumes are most often given off in the

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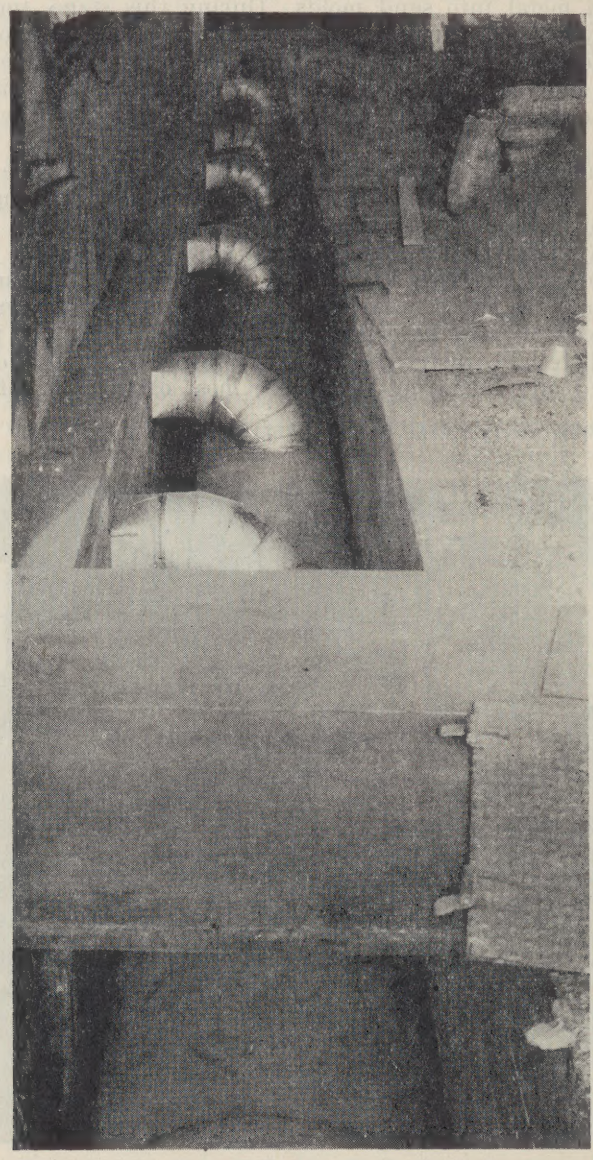
**Pouring Station of Mold Conveyor**

**The weight shifter, who may be seen to the left of the pourer, is the person most exposed to carbon monoxide gas in foundaries.**

ovens when the cores are dried. This process usually takes about 12 hours. In one plant where cores were dried for only three hours it was possible to detect acrolein in the air at the breathing level of workers during the "gassing stage" following the pouring of the metal.



**Exhaust Duct from Hot Casting Conveyor Tunnel**

Organic materials used as binders in core making are usually present in the sand in the ratio of one to 60 parts of sand. Our laboratory tests have shown that every one of these binders when used with sand in these proportions and heated to about 2700°—the temperature at which molten iron is usually poured—will give off irritating fumes.

In view of the number of organic materials used in conjunction with the sand in foundry work and the high temperatures at which the metals are poured, the question arose as to whether or not carbon monoxide might also be present during the "gassing stage" of the molds.

Air tests were made, to this end, in 12 foundries—during the pouring of ferrous and non-ferrous metals. Carbon monoxide was found in varying amounts as indicated in the following table:

| Plant | Kind of Molding | CO Pts per 10,000 Over Molds | | | | Pouring time |
|---|---|---|---|---|---|---|
| | | 2″ above | | Breathing Level | | |
| | | Immediately | 10 minutes later | Immediately | 10 minutes later | |
| 1......... | Brass | 0.4 | 0 | ....... | ....... | Continuous |
| | Iron | 3.5 | 0.7 | 0.3 | 0.2 | 3 hours |
| 2......... | Brass | 0.2 | 0 | ....... | ....... | Continuous |
| 3......... | Iron | 8.0 | 6.1 | 4.5 | 2.2 | 5 hours |
| 4**....... | Iron | 4.5 | 2.0 | 1.8 | 1.0 | 6 hours |
| 5......... | Iron | 6.1 | 3.0 | 2.6 | 1.2 | 3 hours |
| 6*........ | Iron | 1.4 | 0.2 | 0.6 | 0.2 | 3 hours |
| 7......... | Iron | 3.6 | 2.1 | 1.0 | 0.4 | 2.5 hours |
| 8......... | Iron | 2.8 | 1.1 | 1.0 | 0.7 | 6 hours |
| 9......... | Iron | 3.6 | 1.2 | 1.3 | 0.8 | 2 hours |
| 10......... | Brass | 0.8 | ....... | 0.2 | ....... | Continuous |
| 11**....... | Iron | ....... | ....... | 3.0 | 1.0 | 2–8 hours |
| 12......... | Iron | 3.8 | 2.6 | 10.8 | 0.6 | 1.5 hours |
| | Iron | over 15 | 5.0 | Two large molds with pitch) | | |
| | Brass | None | No cores used | | | |

* This plant does not use any sea coal.
** Mold conveyor used in pouring operations.

The production of carbon monoxide was definitely traced to three main sources:

1 Pitch (powdered coal tar residue) used in dry sand molding.
2 Sea coal (a finely powdered soft coal) used as a facing.
3 The organic core binding materials already discussed.

The pitch gave off the greatest amounts of carbon monoxide gas; the sea coal somewhat less, and only relatively small amounts were released from the core binding materials. Tests made on molds which do not contain any of these materials gave negative results.

Practically all of the carbon monoxide is released during the "gassing state" of the mold over a period of from five to 10 minutes immediately after pouring.

The extent to which individual workers may be exposed to carbon monoxide depends largely upon the way in which the process is carried out in the particular plant; the size and number of castings; the number of hours per day during which the pouring is done, as well as upon whether the pouring is continual or intermittent. Of all the workers who take part in the pouring operations, the weight-shifters are the ones who are most exposed to the carbon monoxide gas, because they follow on the heels of the man who pours the metal, and are apt to be in close proximity to the mold when the gases are given off.

When there are only a few large molds distributed over a large floor area, the workers tend to leave the aisles near the hot molds as soon as possible after the pouring has been completed, and are therefore in the vicinity of the "gassing" molds for only a few minutes at most. However, when there are a great many small castings to be made, and the molds are close together, either on the floor or elsewhere, there is less opportunity to walk away from a given mold when it is "gassing," and exposure to carbon monoxide as well as smoke and other irritating gases is more continuous, and therefore greater.

Where a mold conveyor is used, it is the custom for the weight-shifter to stand in one place and do his work on each mold as it is brought to him. Such a worker is subjected to constant exposure to the higher concentrations of carbon monoxide gas. He is stationed only a few feet from where the pouring operation is done, and the molds pass in front of him in the "gassing" state. In removing the weights he is required to bend over the molds, which brings his face about two feet from the top, and very close indeed to the fumes as they are given off.

## Recommendations

1. Where there is excessive exposure to carbon monoxide, smoke or irritating gases during the "gassing" of molds following pouring operations, adequate provisions should be made for the removal of all such noxious fumes.

2. Where conveyors are used for carrying molds to be poured, weight shifters should be especially protected because of their continual proximity to the gases which are given off.

3. Cores, which when hot, give off irritating fumes should not be removed from the core ovens until they are thoroughly cooled off. If, due to the nature of the working conditions this is impracticable, other core binding materials of less irritating nature should be substituted.

This study will be continued, and a more comprehensive report presented in the near future.

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**Photographs by Fred H. Stebbins, Mechanical Engineer, Division of Industrial Hygiene.**